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Original Contributions

New Techniques in Cholecystography

L. HENRY GARLAND, M.B., B.Ch.
San Francisco, California

THIS is the thirtieth anniversary of the death of the distinguished clinician in whose honor this lecture is named. It happens also to be the thirtieth anniversary of my own induction into training in the specialty to which he, Russell Carman, added such lustre. Need I say how deeply honored I feel to have been invited here by the Minnesota Radiological Society (which sponsors this lecture at the annual meeting of your state association), especially since the city in which it is being delivered is that wherein Dr. Carman's own life work reached fruition?

To speak on any radiological subject in Rochester is akin to bringing carboniferous material to Newcastle. And yet, having accepted your invitation, I must try. To speak on the subject suggested by your Committee on Scientific Assembly, namely, cholecystography or cholangiography, is tantamount to effrontery, since Carman's successor, Dr. B. R. Kirklin, is a world authority thereon. But your committee has a persuasive chairman, and I shall therefore assay a brief discussion of certain aspects of the field of cholic radiology.

* * *

When I commenced my residency at Stanford in 1926 (under Dr. W. E. Chamberlain), nonopaque disorders of the gall bladder were diagnosed partly by evidence of displacement or indentation of adjacent viscera (by an enlarged vesicle), and partly by signs of "reflex irritation." The diagnostic reliability of these indirect signs was known to be of low order. Then, like a beam of sunlight into a darkened cave, came the availability of a discovery made two years previously by colleagues of Carman working in the city which he had left about 1914 to join the Clinic.

It had been known for several years that certain dyes were excreted almost entirely by the liver in the bile. Amongst these was tetrachlorphenolphthalein, which was used as a liver function test.

The Carman Memorial Lecture, delivered at the annual meeting of the Minnesota State Medical Association, Rochester, Minnesota, May 21, 1956.

In 1924, Graham, Cole and Copher of St. Louis[3] substituted bromine for the chlorine in this molecule and showed that after intravenous injection enough of the opaque material entered the gall bladder to produce a roentgen shadow. The reactions following intravenous bromph-phthalein were rather severe and iodine was substituted. Later it was discovered that after ingestion of tetraiodophenolphthalein the dye was absorbed from the intestinal tract, excreted in the bile and a gall-bladder shadow produced. Dr. Warren Cole, now at Chicago, kindly gave me copies of slides showing the first cholecystogram obtained in a dog, and the first in a human subject. He also furnished this historic photograph of the three research workers made outside the Mallinkrodt Institute some ten years after their discovery. (Slides shown.)

The water absorptive power of the gall-bladder mucosa plays an important part in the production of the orally obtained gall-bladder shadow. Unconcentrated bile directly from the liver does not usually contain enough of an orally administered medium to cast a good shadow. In the gall bladder, water is absorbed from the bile and the amount of solids (and therefore of opaque elements) is increased approximately fivefold. The result is a relatively dense shadow produced by the contents but commonly referred to as the gall bladder.

Since 1930 numerous and improved media have been developed in various parts of the world, all of them tending to reduce the incidence of undesirable reactions and improve the opacity of the roentgen shadow. Indeed, the quality of some of these media is such that shadows of too dense nature may easily be produced and result in the masking of calculi and papillomata.

There is a vast amount of literature on the need for certain types of diet before and after

cholecystography; indeed, several pages are devoted to the topic of different menus in one excellent loose-leaf diagnostic roentgenology. It is now apparent that many of these dietetic rituals are not essential. However, as in the examination of any abdominal structure, reasonable colonic cleansing and reasonable absence of confusing objects such as buttons and folds of clothing are aids to improved roentgenograms.

Preliminary Examination

Because many of you are general practitioners, I would like briefly to re-emphasize and illustrate the great value of the properly made "plain film of the abdomen" in patients suspect of gall-bladder disease. The following is a summary of the more important conditions it may disclose—many of which will obviate the need for subsequent biliary opacification.

1. Opaque biliary calculi. To detect some of these, we should recollect the wide variation in location of the gall bladder, in both horizontal and erect positions. *Biliary opacities,* including limey bile, may be masked by spine or pelvic bone shadows. The value of a cleared colon, arrested motion and *adequately large* survey roentgenograms is considerable.

When differentiation between biliary and other calculi is difficult, or when identification of probable localization is desired, anterior stereoscopic, lateral or oblique projections are of considerable value. The former have fallen into undeserved disuse.

2. Non-opaque calculi. Cholesterin stones occasionally contain stellate gas shadows to reveal their presence in plain films.

3. Biliary wall disease. The gall-bladder wall may be visible because of *calcification* or *gas.* Calcareous cholecystitis is not common. Emphysematous cholecystitis is even more rare, but readily diagnosable. The gas may be luminal and ductal, as well as mural.

4. Biliary tract air or opaque media. The contrast medium may be vesicular or ductal (and intra- or extra-hepatic); the culprit calculus or ulceration is often difficult to locate. Most cases of spontaneous *internal biliary fistula* are due to gallstone erosion into the gastrointestinal tract.

5. Pancreatic calculi and calcifications. These may project in the gall-bladder area on oblique roentgenograms; their differentiation is based on size, shape and location; *supine and lateral* films should supplement the prone views.

6. Miscellaneous opacities. These include medicinal substances or contrast media in the colon (we have seen pills and barium residues in diverticula misinterpreted as gallstones on occasion); calcified mesenteric nodes; renal calculi and calcifications; arterial calcifications; *adrenal* calcifications; dental and osseous material in *dermoids;* extrinsic and cutaneous opacities. Hydrops may sometimes be demonstrable.

By careful examination and consultation most of the above opacities or lucencies can be correctly localized and identified. When no pertinent abnormality is detected on the plain films, contrast study may be indicated.

Cholecystography

The simplest, safest and most effective general method of cholecystography is by the oral administration of contrast media[2,4,8,9]. Satisfactory media currently available are iopanoic acid (Telepaque) and iodophenoxic acid (Teridax)[10]. Our standard procedure for the average adult is as follows:

1. The patient is requested to have his regular evening meal at about 6:00 p.m. At 7:00 p.m. he takes 2 gm. (four tablets) of Telepaque, and refrains from food and liquids thereafter.

2. At 9:00 a.m. a survey film is made: if a satisfactory shadow of the gall-bladder contents is obtained, two or more additional films are made at different phases of respiration or in different positions (erect and right lateral decubitus), in order to detect any abnormal shadows that may be present. In selected cases, multiple erect "spot films" are made with the fluoroscope, using graded compression, especially for the study of mural radiolucencies. The patient is then given breakfast or its equivalent (a good emptying lipid preparation being Neocholex), and, depending on the problem at hand, films made ten to thirty minutes later. The earlier films show the ducts; the later films, the partly contracted gall bladder. The ducts can be seen in about 75 per cent of standard examinations.

3. If the initial films show a faint or absent gall-bladder shadow, the patient is instructed to repeat the medication again at 7:00 p.m. If he is large (over 165 pounds) the dose is doubled. He is examined next morning. If a satisfactory shadow is obtained, the examination is continued as indicated above. If no shadow is obtained, the examination is terminated and reported as "non-functioning gall bladder on two successive oral examinations."

The above routine presupposes several things: (1) that the patient is able to co-operate, take the medicine at the correct time, and subsequently fast; (2) that he has no pyloric obstruction or marked intestinal hypermotility; either may prevent adequate absorption of iodine; (3) that his colon is reasonably clean; if not, he is given a cleansing enema by the x-ray technician at the time the initial cholecystogram is made. We have tried and abandoned other methods (such as parenteral pitressin) for gas dispersion. For patients who will not co-operate, or have pyloric obstruction, the intravenous method is used.

In private practice, dealing with ambulatory patients and with skilled x-ray technicians, the percentage of satisfactory examinations is very high, exceeding 99 per cent. In county hospital or clinic practice, expecially with bed-confined patients, or with less interested technical help, the percentage is lower. Side-reactions, in either group, are negligible. Seedorf and others have reported transitory, silent, pseudoalbuminuria in about 15 per cent of patients. A variable number complain of mild dysuria (not related to the "albumin" excretion).

The value of immediate repeat examination is evident from the following data, based on the last several hundred cases seen in our office:

The standard (2 gm.) single dose examination is diagnostic in about 90 per cent of cases: the gall bladder shows fair to normal function, and stones (if present) are demonstrated.

Repeat oral examination (using single or double dose, depending on the patient's size) is diagnostic in the remaining 10 per cent of cases. The distribution of these repeat studies showed the following approximate figures:

Normal function, without stones, 2 per cent.
Normal function, with stones, 2 per cent.
Poor function, with stones, 3 per cent.
No function or miscellaneous lesions, 3 per cent.

It is our opinion (based on the cases we have examined) that intravenous examination subsequent to a non-functioning initial oral study has seldom been more informative than a repeat oral examination as far as cholecystic disease is concerned. However, if one is dealing with patients unsuitable for, or unco-operative with, oral study, the intravenous route may be indicated. It sometimes reveals common-duct stones not shown on the oral films (Fig. 1). It may be helpful in jaundiced patients.

TABLE I. DIFFERENTIAL DIAGNOSIS OF "TUMORS" IN FUNCTIONING GALL BLADDER

In erect spot films of gall bladder, without or with compression.

	Papilloma	*Cholesterol polyp*
Size (diam.)	5-10 mm.	1-5 mm.
Shape	Irregular	Round or oval
Number	Single (50%)	Single (66%)
Significance	Potentially neoplastic	Metabolic disorder?
Approx. Incidence in G. B. Studies	0.5%	0.5%

After Hammer, R. W.*

The reliability of properly performed cholecystography for the detection of cholesterin calculi in a functioning gall bladder is almost 100 per cent. For the detection of cholecystitis it has a closely related figure, so long as the technique is correct and some moderate degree of liver function is present. A "nonfunctioning gall bladder," confirmed by an immediate repeat or second examination is indicative of biliary tract disease. Kirklin and O'Donnell[4] reported that in patients having surgery after cholecystography, 98 per cent of positive diagnoses were confirmed. Wickbom and Rentzhog[12] found a similar figure. Moes[6] recently reported the accuracy of cholecystography in 200 surgically verified cases as about 98 per cent.

At the same time, it must be remembered that failure to demonstrate stones does not always mean that small cholesterin calculi can be excluded. Conversely, a normally concentrating gall bladder may sometimes be partly diseased. It is estimated that if one third of the normal gall-bladder mucosa is present, fairly good concentration can sometimes be achieved. Many authors have commented on the excellence of some gall-bladder shadows despite microscopic evidence interpreted as chronic cholecystitis.

The most common sources of error in cholecystography are poor preparation of the patient, and incorrect roentgen examination (both technical and interpretative).

Space does not permit the illustration of the many anomalous and pathologic conditions of the biliary tract demonstrable with cholecystography. However, mention should be made of the increasing demonstrability of lesions formerly difficult to show. One of these is diverticulosis of the gall bladder, also known as Luschka's crypts,

and Rokitansky-Aschoff sinuses. The latter are not uncommon in gross specimens of chronic cholecystitis but were seldom demonstrated radiologically.[7]

Intravenous Cholecystography

Since the introduction of sodium iodipamide (Cholografin or biligrafin) in 1952, there has been a recrudescence of interest in intravenous cholecystography. Indeed, some workers have proposed the "rapid" intravenous method as being preferable to the oral one.[1]

The indications for intravenous cholecystography may be summarized as follows:

1. Persons with serious intestinal tract disturbances such as pyloric obstruction, intractable vomiting, etc.
2. Unreliable or unco-operative persons, including perhaps infants.
3. Occasional patients with acute abdominal conditions in which emergency examination of the biliary tract is indicated.
4. Patients with persistent biliary symptoms following operation (including those in whom the nature of the operation was uncertain, those with postcholecystectomy syndrome, and others).
5. Exceptional cases in which the gall bladder has failed to show function after two properly performed consecutive oral examinations. (In most such cases, the correct diagnosis is gall-bladder disease, and the intravenous method is not necessary.)

The reasons that more general use of the intravenous method is not regarded as advisable include the following: the hazards are greater than with the oral method; there are about 5 per cent of disturbing side reactions with intravenous cholecystography as compared to almost none with oral cholecystography.[8,11,13] The time required in the x-ray department is considerably longer, and the cost of materials is greater. The gall-bladder shadows are confused by associated pyelographic shadows in about 15 per cent of cases. (This does not necessarily indicate liver damage.) Finally, and most important, the films may be misleading if the gall-bladder bile is not well mixed. Prone films made early in the examination may disclose a relatively dense peripheral shadow in the gall-bladder area, presumably due to more concentrated opaque bile close to the water-absorbing mucosa; the central radiolucent area may resemble a large cholesterin stone. Films made with a horizontal beam may disclose stratification or layering of the gall-bladder shadow resembling or concealing floating stones. These layers apparently represent bile of different specific gravities, the lowest being concentrated opaque bile, the intermediate zone, some of the nonopaque bile originally in the gall bladder, and the upper zone unconcentrated opacified bile entering from the ducts.

Aldridge[1] uses the following method for intravenous cholecystography: The patient is prepared by having him take a meal or suitable fatty preparation about two hours prior to reporting for examination; this helps to obtain an empty gall bladder. When preliminary films disclose the necessity therefor, a cleansing enema is given. In the average adult, 50 mg. of demerol is then injected intravenously, in order to increase the tonus of the choledochal sphincter and aid retention in the ducts and gall bladder. After flushing the needle thoroughly (to prevent precipitation of the opaque medium), 10 cc. of 40 per cent Cholografin is injected very slowly; a larger dose is used for obese patients. Films are made about forty-five minutes after injection; if satisfactory gall-bladder shadows are obtained, the patient is then given the usual emptying meal and contraction films made after about twenty minutes. Aldridge reports that he was successful in demonstrating a gall-bladder shadow in a high percentage of 100 patients, including three of eleven jaundiced cases. This latter finding may, of course, mean that opaque ductal bile merely backed up into the gall bladder, not that the gall-bladder mucosa was capable of concentrating it.

Undesirable reactions to intravenous Cholografin include nausea, vomiting, occasional sensation of burning in the throat or flushing of the face and nose, itching, sneezing and transitory cutaneous wheals. There is no satisfactory preliminary test by which such reactions may be predicted. We have seen one case of cutaneous necrosis following a preliminary skin test. Probably the best method of diminishing or reducing reactions is slow injection and suitable preliminary medication in the case of persons with a history of allergy.

Non-operative Cholangiography

Since the subject of cholangiography is of considerable interest at the present time, a few words on this topic may not be out of place. In patients

with functioning gall bladders, *"oral"* cholangiograms may be secured in as high as 90 per cent of cases by correct timing of the after-breakfast gall-bladder films. When films are made approximately ten minutes after an emptying or fatty meal, the cystic and common ducts are usually visible. In some patients, the main hepatic ducts are also visible.

The important place of cholangiography is of course in persons with a "post-cholecystectomy" syndrome in whom there is a question of residual stones or an obstructive lesion.[5] We have secured satisfactory cholangiograms in a fair percentage of cases by the oral method and in a somewhat higher percentage of cases by the intravenous method.

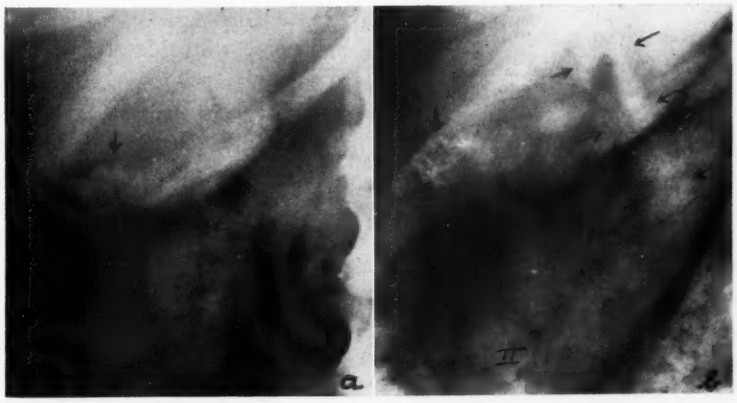

Fig. 1. Preoperative demonstration of common duct stones. Example of occasional value of intravenous opaque medium supplemental to oral examina- duct). (b) Repeat oral examination next day confirms a non-functioning gall cholecystogram (there are multiple calcified stones in the gall-bladder; there is no visible gall-bladder function; there is a faintly visible dilated common duct). (b) Repeat oral examination next day confirms a non-functioning gall bladder and a dilated common duct (17 mm.). However, the contrast in the latter is not sufficiently good to rule out stones.

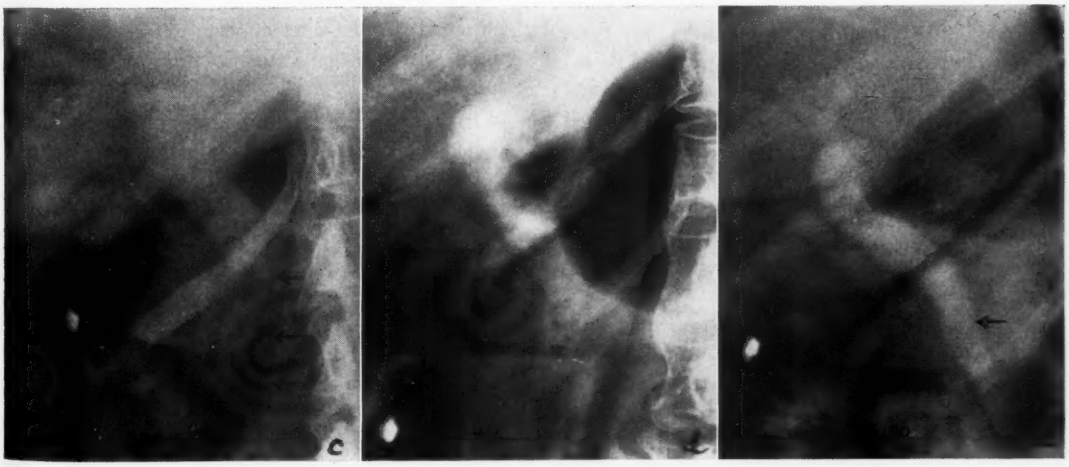

Fig. 1. (c) Examination after intravenous cholografin. There is excellent visibility of the hepatic ducts and the dilated common duct; multiple cholesterin stones ranging from 5 to 15 mm. in diameter are seen in the distal half of the common duct. The timing of these films (c, d, and e) was thirty, forty and 120 minutes after injection.

TABLE II. DIAMETER OF COMMON DUCT SHADOW (IN ROENTGENOGRAMS OF ADULTS)

	Mean	Range
1. "Normal" subjects (no operation)	5 mm.	(3-10)
2. Post-cholecystectomy (no symptoms)	8 mm.	(3-15)
3. Post-cholecystectomy "syndrome"	11 mm.	(5-25)

The following is our technique for the oral method. The patient is given a double dose of Telepaque at 9:00 p.m. (4 grams for the average adult). At 11:00 p.m. he is asked to take 2 drams of paregoric and to refrain from other liquids or food. The initial films are made approximately ten hours later. In thin patients and with correct radiographic technique, the common duct can be shown in about 50 per cent of cases, especially those with obstructive disorders.

The intravenous technique is similar to that previously outlined for intravenous cholecystography. Depending on the problem at hand, the patient may or may not be given a small dose of demerol or morphine prior to the examination. He is then given 20 cc. of 40 per cent Cholografin intravenously, the injection being made very slowly during a period of about eight minutes. Roentgenograms of the biliary area are made at about ten, twenty and thirty minutes. When the duct visualizes slowly, or the presence of stones or stricture is uncertain, additional roentgenograms are made at sixty and 120 minutes. In cases where stones are clearly visible in the initial roentgenogram, and in which this is the only diagnostic problem, the examination may be terminated therewith.

In a series of some 300 patients with symptoms referable to the biliary tract (one-third of whom had not been operated upon), Wise and O'Brien[13] obtained ductal shadows by intravenous cholangiography in about 85 per cent. They believe that non-visualization is apt to occur only "if the serum bilirubin value is above 4.8 mg. or the sulfobromophthalein retention is above 40 per cent."

Berk et al in a smaller series found the ducts to be demonstrable with oral examination (Telepaque) in 55 per cent of patients, and with intravenous medium (Cholografin) in about 90 per cent.

The diameter of the common duct as it appears on the roentgenograms is influenced by many factors, including the thickness of the patient, the target-film distance, the tonus of the duct and choledochal sphincter, and the intraductal biliary pressure. The roentgen image is one usually magnified about 30 per cent. The common duct is often wider after cholecystectomy. Further, the mean diameter tends to be somewhat higher in patients with the so-called postcholecystectomy syndrome than in those without symptoms of recurrent biliary tract disease. In Table II are representative figures for adults (based on our own experience, plus that of Berk et al, Shehadi, and Wise et al).

It has been suggested by Wise and O'Brien that ducts less than 8 mm. in diameter are probably not obstructed, while those more than 15 mm. probably are partly or completely blocked. They utilize the *persistence of a dense ductal shadow* for two or more hours as suggesting obstruction in ducts measuring between 8 and 15 mm. In our experience, early appearance of the duct shadow and its gradual fading away during a two-hour period suggests normalcy; late appearance and persistence of the shadow suggests obstruction. Further, fading out of the terminal portion of the common duct shadow tends to be normal, while a blunt or cigar-shaped terminal duct tends to indicate obstruction. These observations require avoidance of sphincter tonic medication such as demerol, morphine, et cetera.

The apparent length of the common duct should be noted: if the duct appears to be shorter than usual, stricture or extrinsic pressure should be suspected.

In addition to the problems previously mentioned (layering of bile and superimposed pyelogram shadows) an occasional source of confusion in the interpretation of cholangiograms is the presence of opaque medium in the duodenal bulb or gastric antrum; the opacified bile trickles into the duodenum in the recumbent position and, by gravity or reverse peristalsis, enters the duodenal bulb or gastric antrum, with subsequent production of a pseudo-gall-bladder or pseudo-diverticulum shadow. It should be repeated that with either oral or intravenous cholangiography, the failure to demonstrate ductal calculi does not exclude their presence. Examination of the films should be performed with good illumination, multiple views and, when possible, joint consultation between the referring physician and the radiologist. Indeed, "two pairs of expert eyes" are often revelatory in the interpretation of the faint or delicate shadows sometimes existing in non-operative cholangiograms.

Summary

Cholecystography, thanks in part to continuous chemical research and in part to improved roentgenologic technique, is undergoing steady improvement.

The importance of proper preparation of the patient, correct dosage of opaque medium, regular sequences of roentgenographic exposures, and good roentgenologic technique is again stressed. To these, I would add critical examination of the roentgenograms under optimum conditions of trans-illumination, and joint consultation.

The shadows of the biliary ducts are visible in a high percentage of post-cibum oral cholecystograms; attention to such shadows will permit the *preoperative* diagnosis of ductal stones and deformities more frequently than hitherto. When in doubt, immediate intravenous iodixamide may be used to supplement the oral study.

Intravenous cholecystography is indicated in a very small percentage of patients, notably those with obstructive upper gastrointestinal lesions, moderate degrees of jaundice, or situational inability to co-operate with simpler and safer oral examinations.

The great usefulness of nonsurgical cholangiography is stressed, especially in patients presenting diagnostic problems relative to the common bile duct.

Roentgen conclusions concerning apparent disease involving the terminal portions of this duct ("stenosing papillitis," "sphincteric fibrosis," et cetera) should be tempered by complete information as to the patient's concurrent medication. Drugs augmenting choledochal sphincter tone may produce transitory defects.

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X-RAY AS AN AID TO PINPOINTING NEUROMUSCULAR DISEASE EARLY

The importance of the roentgen examination has given the pediatrician and radiologist the opportunity of being among the first to suspect the diagnosis in neuromuscular disease of the urinary tract in children, according to R. Parker Allen, M.D., whose report was published in the September, 1955 issue of *Radiology*.

"Neuromuscular disease of the urinary tract results from the variety of congenital and acquired conditions. It is usually complicated by infection and obstruction. Because of the lack of clearly defined diagnostic criteria, it is often neglected in differential considerations," Dr. Allen explained.

Some other conclusions advanced by Dr. Allen:

. . . Many of the disorders of the urinary tract in children have some neurogenic basis. Those with definite associated clinical disease present practically no diagnostic problem except for determination of how much of the difficulty is due to the neurological disorder and how much to subsequent infection and obstruction.

. . . Cases associated with megacolon are more easily detected because of the known association of the two conditions and the similarity of pattern in cases already reported.

. . . The remaining group of neurogenic dysfunctions, unassociated with other disease, must be differentiated from the much more numerous mechanical obstructions and associated chronic infections. Cystograms, pyelograms, cystometrograms, and in more difficult cases medical and surgical therapeutic trials, are the basis of differentiation.

Posttraumatic Intraocular Hemorrhage

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IT IS encouraging to realize that at last we are becoming properly conscious of posttraumatic intraocular hemorrhage, a condition that is capable of robbing a patient of useful vision in the affected eye. Perhaps our awareness has been sharpened by war experiences, which demonstrated that intraocular hemorrhage is a common finding in concussion injuries from blast; or perhaps it is just that more of us have had to see a young girl or boy lose the sight in an eye because a form of treatment that would unfailingly prevent this catastrophe has not yet been devised. Admittedly, few individuals suffer a loss of vision by this route, but the low incidence neither lessens the tragedy nor relieves us of our obligation to find a mode of treatment sufficiently effective to promise at least reasonably good final visual acuity for all these patients.

In civilian life children and teenagers are most often the victims of direct trauma to the eye—from stones, BB shots, tennis balls and the like—and, as might be expected, the injury is usually sustained during recreational or sports activities. Among adults, the injury is also likely to occur in recreational as well as occupational pursuits.

Intraocular hemorrhage may be classified as:

I. Anterior chamber hemorrhage.
   1. Single.
   2. Secondary or recurrent (and according to Chandler:)
      (a) Iris invisible, blood clot black with surface homogeneous.
      (b) Iris partially visible, blood red with structure of hemorrhage distinguishable.

II. Vitreous hemorrhage
   1. Single
   2. Secondary

We are concerned here primarily with anterior chamber hemorrhage and the emphasis will be on secondary and recurrent anterior chamber hemorrhage since it is principally in patients experiencing two or more hemorrhages that we must fear the development of complications that can lead to a partial or complete loss of sight.

At this point let us review the pathological anatomy in intraocular hemorrhage. According to O'Neill it may be: (1) a tear at the root of the iris, (2) a rent in the ciliary body, (3) the forcing of blood back through the canal of Schlemm.

Cordes and Horner correlate certain features of the lesion with the pathological anatomy and say:

1. Rapid bleeding into the anterior chamber can follow a rupture of Schlemm's canal, the rupture being caused by a small rounded object striking the corneoscleral margin.

2. Rapidly *absorbing* anterior chamber hemorrhage usually follows injuries limited to the iris—(such as iridodialysis, rupture of the sphincter, iridemia and radial or circular tears).

3. Hemorrhages *filling* the chamber are produced by layer tears.

4. In injuries of less severity than usual, the blood comes from ruptured vessels and diffuses over the iris.

5. Occasionally, intraocular hemorrhage results from damage to a retinal vessel.

6. *Vitreous* hemorrhages follow rupture of blood vessels of the ciliary body or choroid.

Kilgore's experimental work on monkeys revealed that contusion frequently caused ciliary body damage, particularly anteriorly, and that in these instances separation of the ciliary body from the sclera always occurred—damage ranging from a slight tear of its substance to complete dislocation. His work also suggested that the bleeding noted two or three days after iridodialysis probably is a result of seepage from this impaired tissue.

In concussion injuries from blast, intraocular hemorrhage from rupture of the iris and ciliary body is common.

Not infrequently an eye injured by direct trauma shows only intraocular hemorrhage—in the form of free blood that usually fills about one-third of the anterior chamber. Fortunately, in most cases this initial hemorrhage absorbs with reasonable speed, vision soon becomes normal and the patient goes his way little the worse for his

Read at the annual meeting of the Southern Minnesota Medical Association, Albert Lea, September 12, 1955.

experience. However, certain of these patients—just when they are recovering satisfactorily, are free of pain and have regained fair vision—suddenly develop a secondary hemorrhage, most commonly into the anterior chamber, though occasionally into the vitreous. This secondary hemorrhage may occur any time from the *first* to the *seventh* day after injury but the most likely days are the second, third and fourth.

More often than not this secondary hemorrhage is accompanied by a rise in the intraocular pressure. Of course the early posttraumatic stage may be characterized by an instability of tension but this situation is of little consequence since stabilization usually takes place within a fairly short time. According to Duke-Elder, ". . . the variations in (intraocular) pressure after a contusion are caused by local neurovascular reactions. . . ." However, he goes on to say, ". . . traumatic glaucoma is based on these reactions. Simple hypertony or hypotony is the physiologic response to trauma—glaucoma is the pathologic effect". We are at a distinct disadvantage in the matter of this secondary tension rise because we cannot predict when or in whom it will come, nor can we determine what initiates the rise.

There is disagreement as to whether a clot formation portends a disastrous end result, although there seems little doubt that the eye with free blood in the anterior chamber—rather than a clot—responds more favorably to treatment and less often demands operative intervention.

The question of paracentesis is perhaps less vigorously argued, the consensus being that simple paracentesis is indicated in the presence of increased intraocular tension and frequently indicated in the presence of secondary hemorrhage, particularly if blood fills the entire chamber.

Summing up what our collective experience has taught us to expect in regard to final vision we find that:

1. The occurrence of a secondary hemorrhage always changes the prognosis from good to bad.
2. With an elevation in intraocular pressure, a bad prognosis immediately becomes worse.
3. When secondary hemorrhage develops within forty-eight hours of the time of injury, the ultimate outcome is usually poor.
4. A hemorrhage that takes the form of a tough, rubbery clot often means a rise in tension and additional bleeding.
5. A delay in performing paracentesis—when the indications for it are clear—may spell disaster.

The complications of posttraumatic intraocular hemorrhage, out of which can come the loss of useful vision if not actually the loss of an eye, are: (1) bloodstaining of the cornea, not due to infiltrating blood cells but to absorption of blood pigment, (2) iris atrophy, (3) secondary glaucoma, (4) glaucomatous optic atrophy with anterior staphyloma—occasionally requiring enucleation.

It is Duke-Elder's view that bloodstaining of the cornea—in which, incidentally, there is no bleeding into the cornea but rather a staining caused by absorption of blood pigment through Descemet's membrane from the anterior chamber—is the only *permanent* result that may follow hyphemia. According to Rychener, bloodstaining—macroscopically—ranges in color from a rusty-red-brown, to a greenish-black or yellow, to a white. Ordinarily the staining is disciform with a clear portion of cornea around the limbus so that at certain stages of resolution the appearance may be that of a lens dislocated into the anterior chamber.

Staining clears gradually from the periphery inward, usually leaving a central patch of gray that may clear even after two or more years—*unless* secondary glaucoma is persistent, in which case the cornea becomes white and unsightly. It is the experience of some observers that useful vision is not recovered following bloodstaining and secondary glaucoma *even though the cornea may clear*. Rychener feels that there is an uncomfortably narrow margin between secondary hyphemia and bloodstaining of the cornea and points out that in one patient of his, faint central bloodstaining of the cornea was observed, during paracentesis, only five hours after the appearance of a secondary hemorrhage. Evidence indicates that increased intraocular pressure is the determining factor in the production of bloodstaining of the cornea.

The iris atrophy resulting from hyphemia and increased intraocular tension is an essential type that can, and often does, continue to total aniridia.

It is estimated that secondary glaucoma supervenes in somewhat more than 50 per cent of those cases with secondary hemorrhage and increased intraocular tension. All too often final vision is only light perception, and enucleation is not the rarity it should be. Rones, reporting on non-perforating ocular injuries in soldiers—almost all of

whom showed intraocular hemorrhage and all of whom did require enucleation—lists the following pathologic changes noted in eyes enucleated from one day to two weeks after injury: (1) beginning organization of intraocular hemorrhage, (2) blood-staining of the cornea, (3) inflammation, (4) secondary glaucoma.

In eyes removed from two weeks to three months after injury, the incidence of inflammation and secondary glaucoma was increased and in this group beginning atrophic changes were found. This report serves to point up the relative speed with which these changes take place, thereby emphasizing the importance of devising and adopting a type of initial treatment that will prevent the complications under discussion.

The debate over the use of mydriatics or miotics appears to have been touched off in 1931 when Hardesty, Green and Luedde reported that secondary glaucoma followed almost on the heels of the administration of atropine to a patient with a secondary intraocular hemorrhage. Since that time atropine has slowly lost its popularity, and there is now a widely held conviction that the use of atropine immediately after injury may well be responsible for a subsequent rise in intraocular tension and recurrent hemorrhage.

Rychener, in 1944, urged the use of miotics in place of atropine on the ground that while atropine does dilate the iris and so compresses the iris stroma and the blood vessels it contains, secondary hemorrhages are often caused by injured iris vessels. Physostigmine, on the other hand, constricts the pupil thus allowing the iris vessels to become stretched, a condition that in itself may act to inhibit secondary hemorrhage.

Jackson contends that atropine is definitely contraindicated, his theory being that since a hyphemia is almost bound to be accompanied by engorgement and edema of the uvea, the filtration angle must be kept open, rather than narrowed by atropine, in order to help in the absorption of the blood in the anterior chamber. O'Neill reminds us of a prime disadvantage of atropine—its persistent mydriasis in the event glaucoma supervenes. He also points out that eserine and pilocarpine contract the ciliary muscle, squeezing the blood from these structures and thereby lessening congestion; and perhaps these drugs may also reduce the probability of secondary glaucoma by pulling on the scleral spur and so opening up the canal of Schlemm.

Thygeson and Beard, while not defending atropine specifically, say that since ciliary body damage is a common cause of hyphemia, it would seem wise to use mydriatics as a means of avoiding contraction of the ciliary body, at least for the first five days after injury.

Bair suggests that both miotics and mydriatics are contraindicated because, should continued bleeding be due to an overwhelming vasodilator axon reflex—mediated through the trigeminal fibers and caused directly by contusion—miotics and mydriatics would only augment this vasodilatation by producing a capillary dilatation.

Our failure to reach a decision in the choice of drugs for use in the immediate post injury period stems from our lack of a precise understanding of the mechanisms of hypophemia. Without doubt several factors are involved:

1. The vasodilatation just mentioned.
2. A possible paralysis of the vasoconstrictor mechanism.
3. The hypotony phase of the fluctuating tension found immediately after injury.
4. Perhaps a temporary capillary fragility.
5. And probably others of which we are unaware.

The most important elements of treatment in intraocular hemorrhage appear to be: (1) absolute bedrest for seven days with both eyes bandaged four days, (2) a careful check kept on the intraocular tension, (3) prompt operative intervention when indicated.

In the literature one finds repeated instances of secondary hemorrhage occurring after exertion, frequently when the initial hemorrhage was well cleared. Hence absolute bedrest for a week, the period of time during which secondary hemorrhage is possible, seems imperative. Adequate sedation is also indicated because an agitated, frightened or restless patient is capable of considerable exertion.

The indications for paracentesis are secondary or recurrent hemorrhage and/or increased intraocular tension. If paracentesis must be repeated, as is often the case with recurrent hemorrhage, it is recommended that it be done by inserting a spatula in the anterior chamber through the original paracentesis wound, which is often at the temporal limbus. The old practice of removing a clot with a forceps or by means of lavage has been abandoned by most of us because of the very real danger of injuring the lens through instrumentation. Today the procedure of choice is simple paracentesis.

A little pioneering is now being done in nonoperative treatment and with considerable success. Those who avoid operative intervention do so because there is undeniable evidence that final vision is not as good in the operated case as in the unoperated case. Townes feels that conservatism in treatment is the password to success, particularly with young people. He is of the opinion that the ocular structures of patients up to thirty or thirty-five years of age are sufficiently elastic to withstand increased intraocular tension for prolonged periods without running the danger of irreparable damage or loss of useful vision. His treatment for young people with repeated bleeding and secondary glaucoma is well worth reviewing for he has had remarkable success with it:

1. Absolute bedrest.
2. Adequate sedation.
3. *Infrequent* use of miotics.
4. Pressure bandage of eye.
5. Cold compresses.
6. Vitamin K or cevitanic acid.
7. Intravenous hypertonic solutions.
8. Later, after determining whether vitreous hemorrhage has occurred—intravenous iodides.
9. In the presence of persistent glaucoma after hyphemia has absorbed and ciliary congestion has subsided, the effectiveness of miotics renewed by the *occasional* use of:

(a) Suparenin bitartrate, instilled every ten minutes for one hour, and followed by:

(b) Eserine, instilled every ten minutes for the next hour.

Clark of New Orleans has been extremely successful with a form of treatment that involves a special diet, what he calls a dehydration diet. He uses this treatment, incidentally, in postoperative hemorrhage as well as in initial or posttraumatic intraocular hemorrhage—but he emphasizes that it is not of value in hemorrhage associated with vascular disease.

The treatment begins immediately after injury when the patient is given 50 cc. of a 50 per cent glucose solution by vein, and hospitalized. The following are then ordered:

1. Absolute bedrest, with eyes occluded, for one week.
2. All fluids and foods stopped immediately.
3. Diet started consisting of 2 ounces of white Karo syrup in 3 ounces of fruit juice or water—to be given every three hours during day and every four hours during night, for six or seven days.

No other food or fluid permitted during this period.

4. Ascorbic acid, 100 mg. by hypodermic, twice daily.
5. Appropriate medications for rest and sleep.
6. If bleeding still active, synthetic vitamin K, 5 mg. by hypodermic.

Dr. Clarke points out that he cannot accurately account for the effectiveness of this dehydration regime but suggests that perhaps a sort of reverse osmosis occurs, with the syrup drawing the fluid, and accelerating the elimination of waste products, from the anterior chamber. Or, he offers, it may be that the dextrose acts in some way to prevent further bleeding. In any case, the results of this treatment have ranged from fair to dramatic, with most of the results deserving to be called "good" and many deserving to be classified as "spectacularly good."

The sequelae of postraumatic intraocular hemmorhage are: (1) degeneration, (2) atrophy, (3) lowered viability, (4) lack of biologic resistance to further insult. All of these plead for an open mind in approaching the task of finding a uniformly effective treatment for this lesion.

In my own experience with seventy-two cases the best results have been obtained by adhering to th conservative methods of treatment. Mydriatics and myotics were not used.

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Metastatic Neoplasms of the Penis

Review of the Literature and Report of Two Cases

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METASTATIC neoplasms of the penis are rare. Fifty-six cases were collected from the literature: fifteen originated in the bladder, twelve in the rectum, eleven in the prostate, seven in the kidney, six in the testes, two in the lung, and one each in the liver, bone and nasal pharynx. Route of metastasis is either by the lymphatics accompanying the internal pudendal vessels or hematogenous via the dorsal vein of the penis. The corpus spongiosum and the urethra are not usually involved. Priapism occurs in more than one-half of the cases. The outlook is grave.

Surgical treatment has afforded symptomatic relief in six cases. Cattell and Mace[6] reported a metastatic penile lesion two and one-half years after resection of a carcinoma of the rectum; the patient was living without recurrence nine years after amputation of the penis. Boyd's[4] patient was living without recurrence ten months after radical amputation of the penis, and over five years after excision of a carcinoma of the rectum. The patient reported by Niewiesch had local recurrence and generalized metastases six months after partial amputation. Waller and Hellwig[24] reported no recurrence one year after radical amputation of the penis for metastasis from the prostate. Hamm and Weinberg's[13] patient was living ten months following total amputation and orchiectomy for a metastatic penile lesion of the prostate. A metastatic lymphosarcoma from the testes was treated by partial amputation of the penis; the patient was alive ten months later.

Case Reports

Case 1.—E. F. R. S., a sixty-four-year-old white man, was admitted on July 7, 1952. During the past three years, he noticed a progressive increase in symptoms of prostatism, with nocturia one to three times, intermittency, a weak stream and a feeling of incompletely emptying his bladder. During a routine physical examination at the Cancer Detection Center in May, 1952, a prostatic nodule was discovered; his referring physician placed him on 100 milligrams of stilbestrol daily.

Physical examination was essentially negative except for hypertrophy of the breasts and a small prostate gland with an irregular firm right lateral edge with possible periprostatic extension. The residual urine was 85 cubic centimeters.

The peripheral blood and urine were normal. Acid phosphatase was 1.7 and the alkaline phosphatase was 12.0 King Armstrong units. The blood urea nitrogen was 16 milligrams per cent. Culture of the urine from the bladder was sterile. Roentgenogram of the chest was normal. Roentgenograms of the urinary tract revealed some small diverticula of the colon filled with barium from a previous barium enema, multiple prostatic calculi, and no metastatic lesions of the skeletal system.

On July 11, 1952, cystoscopy revealed a fibrous prostatic collar; the bladder was trabeculated grade II with numerous cellules. Fifteen grams of prostate were resected with a blood loss of 40 cubic centimeters. No prostatic calculi were present in postoperative roentgenograms. The pathologist reported carcinoma of the prostate (Fig. 1). He was placed on stilbestrol 5 milligrams daily.

On September 10, 1954, the acid phosphatase was 1.8 and the alkaline phosphatase was 4.3 King Armstrong units. The right lobe of the prostate was hard and smooth whereas the left presented a sizable nodule which was hard and fixed. Radiographs showed increased density in the left half of the body of the fifth lumbar vertebra as well as a small irregular area of slightly increased density in the left pubis—not at all typical of metastatic lesions. Because of restriction of stream, the stilbestrol was discontinued and Vallestril 20 milligrams was prescribed.

On October 6, 1954, despite the Vallestril, he noticed a further restriction of his stream as well as an ache in the left buttocks which radiated down the posterior thigh and was relieved by moving about. A nodule the size of a No. 2 shot was present at the base of the penis. He was placed on 20 milligrams of stilbestrol daily.

On October 27, 1954, he was admitted, and 27.8 millicuries of radon were inserted into the prostate through perineal needles. The nodule at the base of the penis had more than doubled in size. It was superficial to the tunica albuginea and freely movable, and was excised together with the adjacent tunica albuginea. The pathologist reported metastatic carcinoma of the

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prostate (Fig. 2). On November 23, 1954, four seeds of radon each approximately 1 millicurie were inserted about the periphery of the penile scar. There had been a remarkable reduction in the size of the left lobe of the prostate. Stilbestrol 20 milligrams daily was resumed.

On June 27, 1955, eight months after removel of the nodule, he was voiding sparkling clear urine without difficulty. The breasts were hypertrophied, but not tender; the prostate gland was broad, flat and fixed with some extension over the right seminal vesicle. There was no recurrence of the penile metastases. Stilbestrol therapy was continued.

On August 29, 1955, he was examined because of pain in the lumbar region which radiated along the distribution of the right sciatic nerve. Roentgenograms revealed increased density of the articular facets of the fifth lumbar vertebra and bilateral pulmonary metastases. The acid and alkaline phosphatases were within normal limits. The urine was sparkling clear. The prostate gland was hard and fixed and larger than on the previous examination. There was no recurrence of penile metastasis. Stilbestrol was continued. Irradiation of the lumbar vertebrae afforded relief of his pain.

Case 2.—E. H., a seventy-nine-year-old white man, was admitted on February 25, 1954. For the past year, he had had progressive symptoms of prostatism, culminating in acute retention. Diabetes, well controlled with 30 units of NPH insulin, had been discovered six years earlier. Symptoms of peptic ulcer had been present for the past four years. In 1953, a cerebral vascular accident had resulted in a slight residual weakness of the left arm and leg.

Examination revealed an elderly male with an indwelling Foley catheter. There was a little weakness of the left arm and leg. His blood pressure was 150/70. There were bilateral, lenticular opacities. The heart was enlarged to the left. The prostate was enlarged 2-plus and was hard and fixed, especially on the left; it was very suggestive of carcinoma. The rest of the examination was essentially negative.

The peripheral blood was normal. The urine contained one plus albumin, two plus sugar and three plus red blood cells. The blood urea nitrogen was 15 milligrams per cent; the serum acid phosphatase was 2.1 and the alkaline 4.6 King Armstrong units. The blood sugar was 146 milligrams per cent. A radiogram of the urinary tract did not reveal skeletal metastases; that of the chest disclosed a moderate enlargement of the left ventricle. An old duodenal ulcer with deformity and old sealed perforation was present in the gastrointestinal series. Culture of the urine from the bladder was sterile.

Cystoscopy revealed a Grade I median lobe and Grade II lateral lobe hypertrophy. The bladder was trabeculated one plus. Fifty-one grams of prostate were excised with 160 cubic centimeters blood loss. The pathologist reported benign prostatic hypertrophy. The patient was discharged on March 11, 1954.

On May 10, 1954, he was readmitted. For the past two weeks, he had had chills and fever and a slight

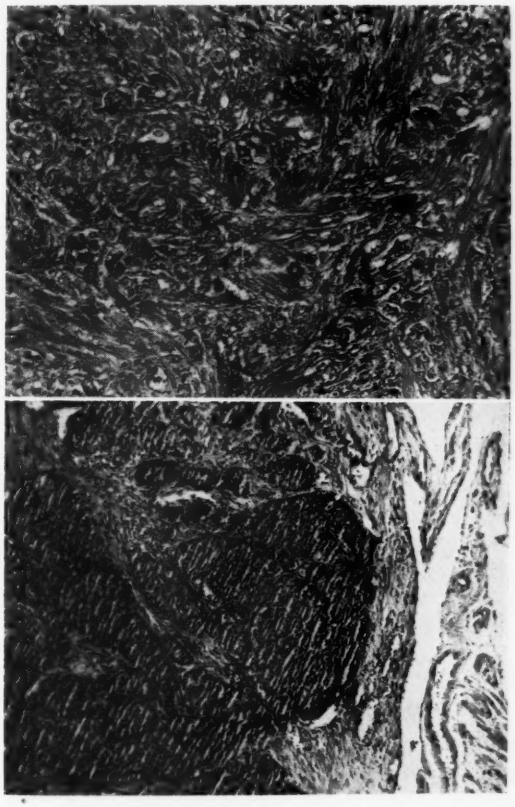


Fig. 1. *(above)* Photomicrograph: carcinoma of the prostate (Case 1).

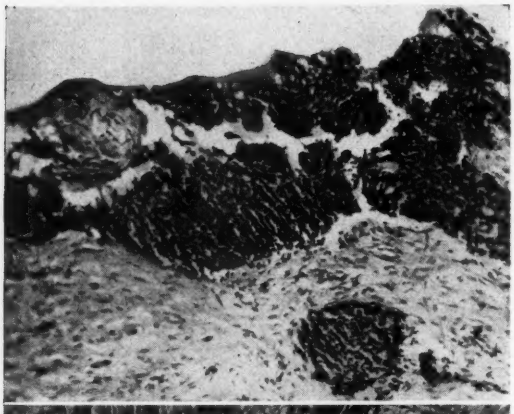
Fig. 2. *(below)* Penile nodule: metastatic carcinoma of the prostate (Case 1).

initial hematuria. He was afebrile on admission. Weakness of upper and lower extremities was still present. The right seminal vesicle was tense, enlarged and tender; the right lobe of the prostate was tense; the left lobe was normal. There was a moderate periurethritis. The rest of the physical examination was negative.

The hemoglobin was 14.6 grams; 8,950 leukocytes with a differential count of 85 per cent neutrophils, 12 per cent lymphocytes, 2 per cent monocytes, and 1 per cent basophils. Urine analysis revealed a specific gravity of 1,026, pH 5, 1-plus albumin, 2-plus sugar and 2-plus white blood cells. Culture of the urine from the bladder contained aerobacter aerogenes. Acid phosphatase was 1.8 and alkaline phosphatase 9.2 King Armstrong units. The excretory urogram revealed no skeletal metastases. The kidneys were normal, but the terminal pelvic ureters were slightly dilated.

Cystoscopy disclosed an excellent resection of the prostate gland. He was discharged on Achromycin.

He was readmitted on October 27, 1954, complaining of increased frequency, initial hematuria, and a gradual diminution in the force and size of his urinary stream, associated with episodes of retention necessi-



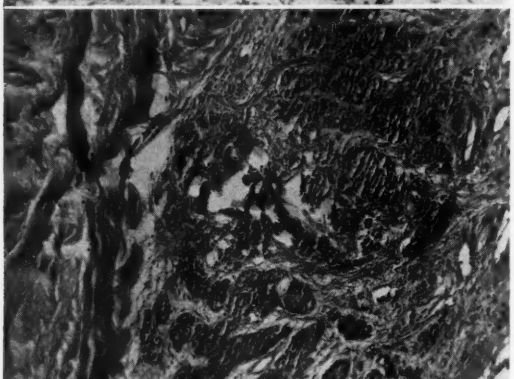


Fig. 3. *(above)* Needle biopsy of the prostate revealing carcinoma (Case 2).

Fig. 4. *(below)* Penile lesion: metastatic carcinoma of the prostate (Case 2).

tating catheterization. There was a pronounced nodular induration along almost the entire ventral aspect of the penis and extending upward in the midportion around the corpora cavernosa. The prostate was hard and fixed. The acid phosphatase was 2.3 and the alkaline phosphatase was 17.4 King Armstrong units. An urethrogram showed a stricture of the midportion of the penile urethra. The prostatic urethra was elongated and there were some small ragged filling defects at its apex. Cystoscopy disclosed necrotic tissue about the apex of the prostate. Biopsies of the prostate and the penis were reported as carcinoma of the prostate with metastasis to the penis (Figs. 3 and 4). A trocar cystostomy and bilateral orchiectomy were done. He was discharged on Stilbestrol, 5 milligrams daily.

On April 4, 1955, he expired elsewhere; the cause of death was hypostatic pneumonia and hemorrhage from a duodenal ulcer. The penile lesion had not increased in size. Necropsy was refused.

Summary

Two additional cases of metastatic carcinoma of the penis from the prostate are reported. This brings the total of secondary malignancies of the penis to fifty-eight.

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(Continued on Page 456)

Familial Absence of Mesenteric Plexus as a Cause of Bowel Obstruction in the Newborn

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THE basic intestinal disturbance in the disease called congenital megacolon, or Hirschsprung's disease, has been clarified in recent years. Papers have appeared reporting abnormalities of the ganglial cells of Auerbach's myenteric plexus of the large bowel. In 1940 Tiffin et al[1] advanced the proposition that the real defect in congenital megacolon was the absence of the myenteric plexus in portions of the colon. This absence of the plexus produced a physiological obstruction. Later, Whitehouse and Kernohan[2] of the Mayo Clinic made careful autopsy studies of eleven cases of congenital megacolon and found the Auerbach plexus missing in the terminal portion of the colon. All of their cases concerned persons who had lived beyond infancy. Swenson[3] and his co-workers extended this knowledge and put it to practical application in the development of the modern surgical treatment for congenital megacolon.

The form of the disease which is seen in ages beyond infancy could be called *chronic* congenital megacolon. The form which presents itself as intestinal obstruction in the newborn, on the other hand, could be called *acute* congenital megacolon and it is with the latter form that we are concerned here. The basic defect in the two forms of the disease, that is, the deficiency in Auerbach's plexus, is the same. The *difference* in the two forms of the disease is that the acute form is more extensive and severe so that the newborn child cannot live without surgical correction. Another apparent difference is that the acute form may be manifest as a familial disease. The literature on chronic congenital megacolon does not stress its occurrence as a familial disease, while the occurrence of the acute form as a familial disease is striking. Zuelzer and Wilson[4] in 1948 reported eleven cases of acute congenital megacolon with obstructive symptoms in the first few days or weeks of life. Five of their cases were siblings of one family. Nine of their patients died early in infancy; two survived with colostomies. Forshall et al[5] in 1951 reported the occurrence of this disease in four siblings, all of whom died in the first days or weeks of life. An aunt of these children probably had congenital megacolon also. Bodian[6] in 1951 reported the occurrence of the disease in both the acute and chronic forms in siblings of three families. As early as 1924 Dalla Valle[7] wrote of two siblings each of whom died in late infancy with obstructive symptoms and with absence of Auerbach's plexus in the distal colon.

The treatment of acute congenital megacolon prior to the past few years was very unsatisfactory. The most common error was to mistake the condition for atresia of the bowel or to blame the obstruction on adhesions. In the latter instance, the surgeon would be misled because the colon would be found to be patent, allowing for the expression of meconium and air. No definitive treatment would be given and the child would usually expire. Occasionally a colostomy would be done, allowing temporary relief. In 1952, Potts[8] emphasized the importance of applying Swenson's technique for the correction of chronic congenital megacolon to the correction of acute congenital megacolon.

Case Reports

The following seven cases, four of them siblings from one family and three of them siblings from another family, illustrate the clinical picture and the familial nature of the disease.

Case 1.—Baby Boy C., weight 9 pounds 3 ounces, was born June 5, 1938, at Swedish Hospital in Minneapolis. There was an onset of vomiting at three days of age. A meconium stool was recorded. An operation was done at eight days of age. The ileum was distended to within two inches of its juncture with the cecum. The obstruction was thought to be due to adhesions at this point. They were released but the child died the same day. At autopsy, no atresia or obstruc-

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tion was found. No microscopic examination of the colon was made.

Case 2.—Baby Boy C., weight 10 pounds 4 ounces, sibling of Case 1, was born on March 28, 1940, at Asbury Hospital. The baby vomited and had a distended abdomen on the second day of life. A barium enema was done (Fig. 1). The baby was operated upon on the third day of life. The ileum appeared to be constricted with congenital bands, which were released. There was free blood in the peritoneal cavity. The baby died the following day. Autopsy showed narrowing of the ileum from the ileocecal valve to a point 15 cm. proximal. The bowel was distended above this point. A microscopic section of the colon is shown (Fig. 2). A section of normal colon is shown for comparison (Fig. 3).

Fig. 1. Case 2. Barium enema showing the narrowed distal colon in the rectosigmoid area.

Case 3.—Baby Girl C., weight 8 pounds ½ ounce, a sibling of Cases 1 and 2, was born on February 23, 1941, at St. Barnabas Hospital. Projectile vomiting occurred on the fourth day. A brown mucoid stool was passed on the eighth day. An x-ray at that time showed an obstructive lesion in the mid-portion of the transverse colon, but barium could be forced beyond this point to the proximal colon. Barium given by mouth passed only to the terminal ileum. An operation on the eighth day showed hemorrhage from the posterior aspect of the cecum and a partial volvulus at the hepatic flexure. No obstructive site could be found; no definitive treatment was done. The baby had a stormy postoperative course and died nineteen days after operation. At autopsy the small bowel was distended and hypertrophied. The cecum was perforated and collapsed. No atresia or area of mechanical obstruction could be found. No sections of the bowel are available.

Case 4.—Baby Girl Frances C., weight 9 pounds 11 ounces, a sibling of Cases 1, 2 and 3, was born on January 17, 1946, at St. Barnabas Hospital. (A different doctor delivered each of the babies.) The abdomen was moderately distended. Meconium was obtained on rectal examination. An x-ray showed distention of the small bowel with no visible air in the colon. At operation on the second day of life, there was distention of the small bowel and ascending colon and the first half of the transverse colon. The fluid contents of the bowel could be expressed into the distal colon. The baby died three days after operation. At autopsy the cecum was perforated; the distal colon was not distended and appeared normal. The single section of the bowel which is available shows no ganglia cells (Fig. 4).

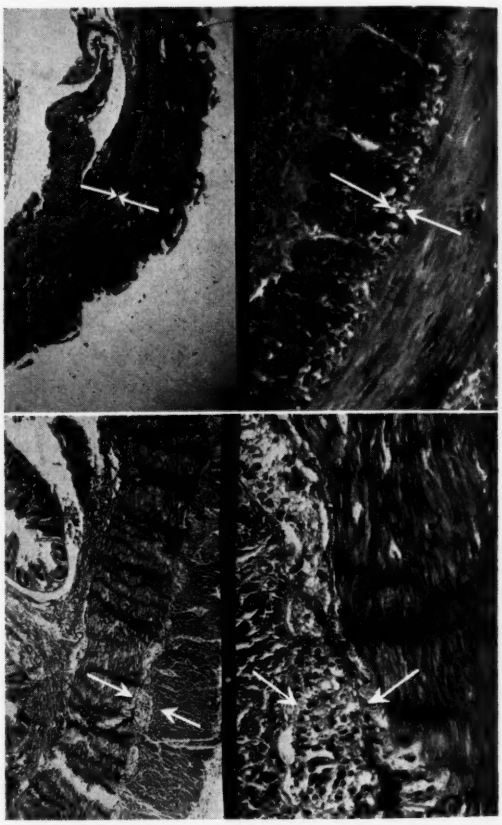

Fig. 2. *(above)* Case 2. Low power view (left) and high power view (right) of section of colon obtained at autopsy. Auerbach's plexus should lie between the longitudinal and circular muscle bundles, but is absent. Compare with Figure 3.

Fig. 3. *(below)* Section of a normal colon from an infant. Auerbach's plexus can be seen as nests of cells lying between the longitudinal and circular muscle bundles on the left (low power). The individual large dark staining cells can be seen in the high power view on the right.

This family then stopped having children. They had one normal daughter born before these babies reported here.

Case 5.—Baby Gregory O. was the fourth child born to his parents. The three previous siblings were normal children with no problem of constipation. There was no history of Hirschsprung's disease or of severe constipation in any relative. This baby was born September 1, 1951, and weighed 8 pounds. There were no stools

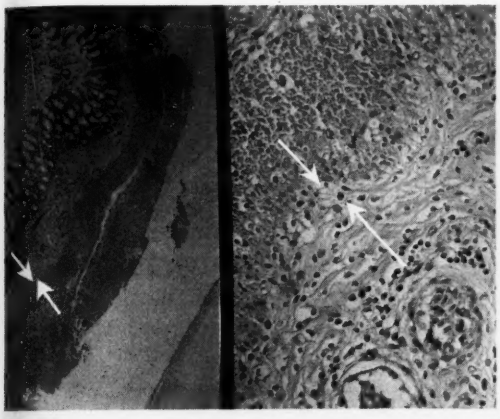

Fig. 4. Case 4. Section of colon showing absence of Auerbach's plexus. Low power on left. High power on right.

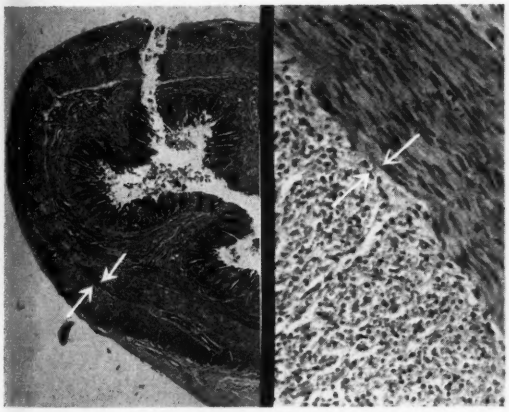

Fig. 6. Case 6. One of serial sections taken from the colon showing absence of Auerbach's plexus. Low power on left. High power on right.

for the first three days, and the abdomen became distended. An enema produced a moderate amount of dark watery stools. A barium enema was done (Fig. 5). Vomiting started on the fourth day. Despite suction the distention persisted. Operation was performed on the eighth day. There appeared to be an obstruction of the jejunum represented by an indentation of the wall of the jejunum. However, when the bowel was opened no septum or atresia was found. The remainder of the bowel, large and small, appeared normal. The postoperative course was good. Stools were passed and weight was gained. The child was discharged at about two weeks of age. After five days at home the child was readmitted in state of shock and dehydration. He died a few hours later. At autopsy a fibrinous dry peritonitis was found. The bowel was distended and necrotic, all matted together. No actual site of obstruction was found.

Case 6.—Scott O., a sibling of Case 5, was born on May 5, 1953. Abdominal distention and vomiting oc-

Fig. 5. Case 5. Barium enema showing narrowed rectosigmoid area.

curred about sixty hours after birth. An x-ray indicated obstruction so operation was done on the third day of life. At operation there appeared to be a herniation of the distal ileum through a rent in the mesentery at the ileo-cecal junction. The entire colon was abnormally small. The herniation was corrected. Some stools were passed about one week postoperatively but on the fifteenth postoperative day the distention was so great that re-exploration was done. Adhesions were found; the small bowel was distended; the colon was still small and empty. An ileostomy was done but the baby died the following day. At autopsy the small bowel was distended; the colon was small—at no point more than 18 mm. in circumference (Fig. 6). Serial sections showed that Auerbach's plexus was missing from the entire colon.

Case 7.—Keith O., a sibling of Cases 5 and 6, was born March 29, 1955. Birth weight was 7 pounds 6 ounces. The child started to vomit soon after birth, the vomiting becoming fecal in nature. No stools had been passed when he was first seen at thirty hours after birth. A rectal examination produced a mucous plug and some meconium, but the abdominal distention was not relieved. A lipiodal enema was done (Fig. 7), showing a narrow distal colon. This was confirmed at operation done on the second day of life. A transverse colostomy was done. The postoperative course was complicated by a staphylococcus septicemia, but the baby is now thriving at ten months of age. He has normal stools through the colostomy, which was fitted with a bag. It is

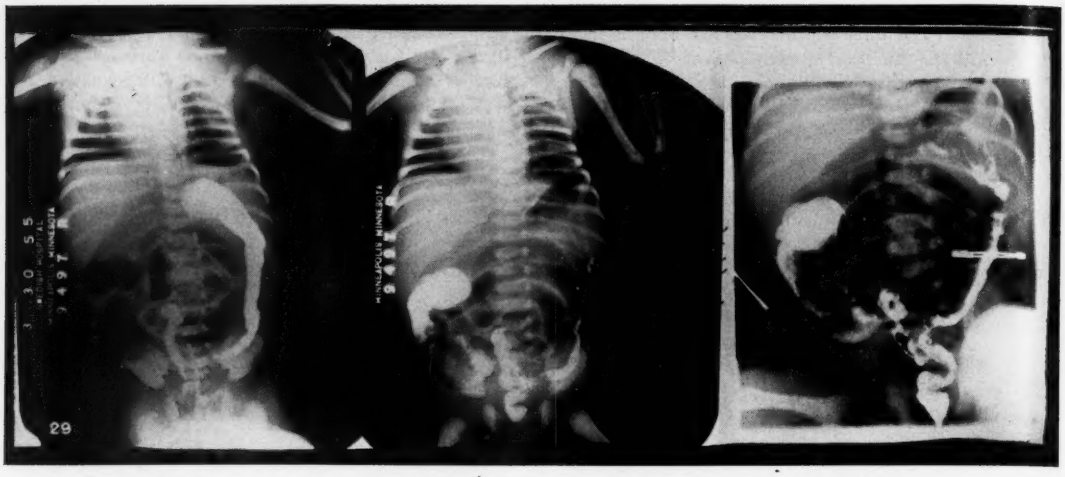

Fig. 7. Case 7. Three views of a lipiodal enema showing the narrowed distal colon. A mid-transverse colostomy has functioned well in this infant.

our plan to do a Swenson pull-through anastomosis at about one year of age. An incidental finding in this baby is a coloboma of the iris of the right eye.

In retrospect, it would appear that three of the babies in the first family might have survived with a colostomy. The others in this series of seven appeared to have all the colon or part of the small bowel involved and probably would not have survived any procedure.

Discussion

We think it is important to emphasize that the syndrome of acute congenital megacolon be considered in any bowel obstruction in the newborn infant. If no other cause for the obstruction can be found, and if a portion of the bowel is dilated and a more distal portion appears narrowed or even normal, then acute congenital megacolon is a sound presumptive explanation.

Potts recommends that a primary anastomosis be done if the baby's condition is good. The narrowed segment, usually distal colon and rectum, should be removed as well as any part of the dilated colon which is deficient in Auerbach's plexus. The help of a pathologist with frozen sections is needed, of course, to determine the extent of surgical excision. If the condition of the baby is not good or if the technical problem appears to be too great at the moment, a transverse or descending colostomy can be done and the final anastomosis done later. The temptation to close the abdomen without a definitive procedure (particularly in the presence of "congenital bands"), in hopes that things will work out, should be resisted. The recorded experience would indicate that such babies will not survive.

Summary

A series of seven cases of congenital megacolon causing bowel obstruction in the newborn infant is reported. Four of the infants were siblings of one family, three of them were siblings of another family. One baby of the seven survived. The importance of recognition of this syndrome is stressed. The pathology and the treatment of acute congenital megacolon are discussed.

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Harmful Effects of Interpretive Psychotherapy in Certain Involutional Depressions

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THE point of departure of this paper is the hypothesis that, broadly speaking, there are two kinds of individual psychotherapy: (1) expressive or deep, and (2) suppressive or supportive therapy.[1] Expressive therapy is designed to explore and elucidate the emotional conflicts that are unknown to the patient but which cause symptoms. It employs analytic techniques such as exploration, abreaction, free association and *interpretation.* Suppressive therapy relies mainly upon ego strengthening measures and employs reassurance, encouragement, explanation of symptoms at a more superficial level, and so forth. Of course, in both kinds of psychotherapy transference plays a very important part.

My thesis is that there are certain depressions in which expressive therapy with interpretation is hazardous and at times even harmful. These depressions are the ones generally known as the involutional depressions. Whether the involutional depressions are endogenous or constitutional—have no external precipitating factors—or whether they are largely reactive—have external precipitating causes such as excessive environmental stress—is debatable. Insofar as an individual can compensate for external threats until he has reached the involutional period of life and that with the onset of the climacteric he may no longer be able to effectuate these compensations, the depression may be said to be reactive.

Depressive emotional responses can occur at various levels, depending upon the inner dynamic structure of the patient's personality. However, I do not subscribe to the Lockean view that the mind is a tabula rasa on which experience writes all that is later to exist. I lean toward the Leibnitzian theory that the mind has an active core of its own. Therefore, even the "reactive" depressions in all probability have an intrinsic factor. All personalities have the capacity to transcend the immediate moment, to make decisions in long-range terms, and they have the elements of freedom and responsibility for ethical living. Consequently, there is no such thing as a purely reactive depression. There always is an endogenous factor.

The first of these endogenous factors is heredity. Franz Kallmann[2] concludes that "the recurring ability to exceed the normal range of emotional responses with extreme but self-limited mood alterations seems to be associated with a specific neurohormonal disturbance which depends on the mutative effect of a single dominant gene with incomplete penetrance. The dynamics identified with this specific ability cannot be considered to be part of a person's normal biological equipment nor are they out of line with current concepts of psychodynamic phenomena as observed in potentially vulnerable persons. Further, the ostensible occurrence of a gene-controlled tendency to excessive mood swings in adults shows evidence of the existence of a regulative mechanism, which ordinarily serves to protect man's emotional life from harmful extremes of affective responses. Obviously, this protective device is also determined by heredity, that is, by the physiological effect of the normal allele of that mutant gene capable of producing the symptomatology of a cyclic psychosis." Later, Kallmann says, "The range of manifest symptoms observable in twin partners and siblings of manic-depressive patients offers an essential criterion for distinguishing the usual symptomatology of manic-depressive psychosis from those of other clinical entities. More specifically, one may say that the understanding of various psychiatric problems related to the etiology of depressive phenomena is likely to improve, if it can be based on a consistent and genetically confirmed system of diagnostic classifications."

The second endogenous factor is endocrine imbalance. Reiss[3] of Bristol, England, reminds us that (1) many ductless glands have a direct influence on the brain metabolism and therefore on different phases of mentation; (2) every psychic trauma or serious psychological disturbance has an

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influence on the function of the ductless glands; (3) it is difficult to think in terms of primary and secondary phenomena when investigating a psychopathologic-endocrine problem since the problem is only confronted when the pathological process is already advanced and a vicious circle has been developed. However, Reiss' work indicates that changes in the total hormone equilibrium show a closer correlation to disturbed mentation than do the changes in the functions of a single gland.

Fabing[4] in discussing the chemotherapy of certain psychoses refers to a number of psychonoxious drugs known to the Germans as the "phantastica" and, more recently, as the "hallucinogens." In his study of these drugs Fabing found that an indole nucleus is common to their chemistry. The substances of vegetable origin include LSD-25 from rye rust, yohimbine, mescaline, and others. There also are hallucinogens of animal origin. These include tryptamine, bufotenene, adenochrome (pink adrenalin), and others. Although these drugs cause schizophrenic-like reactions and do not seem to enter into the production of depressions, in my opinion it is conceivable that other chemical compounds not yet discovered are in some way involved in the causation of depressive reactions. Depressions so caused would, of course, be considered endogenous.

In the involutional depressions we probably are dealing with a combination of endogenous and exogenous factors. The proportion in which each of these factors exists varies with the individual case. However, there is a certain tendency toward polarity, and through a careful evaluation of each patient one should be able to ascertain which factor predominates.

In addition to the usual psychiatric interview, some of the psychometric tests have been very helpful aids in determining what type of psychotherapy should be used in each case. I have found the Rorschach test to be very helpful to both patient and therapist. With this test it is usually possible to ascertain how much of a neurotic (reactive) component and how much of a so-called intrinsic or endogenous component exists in each case. A discussion of the Rorschach variables in this connection does not come within the scope of this paper. Another important psychometric test is the Wechsler-Bellevue. The merit of this test lies in the fact that a knowledge of the amount of intelligence a patient has is necessary before a psychotherapeutic program can be set up. As has been pointed out,[5] "people of dull normal intelligence who are not sophisticated will respond to reassurance and to suggestive therapy, which to a more intelligent person would appear as nothing more than hocus-pocus. By the same token, if a patient has a high normal or superior intelligence, it is possible to treat him with a more penetrating approach." However, when we are dealing with the involutional depressions, an interpretive and penetrating approach may do more harm than good.

I refer especially to those depressions in which retrospective self-accusation is a prominent symptom. Guilt about masturbation and other fancied sexual misadventures should be dealt with in a suppressive way. To explore, analyze, and interpret the dynamics of these guilt reactions in people suffering from involutional depression may mobilize more anxiety than the patient can handle and tend to increase the depression. I am speaking now of the depressions in which the intrinsic factor predominates. A very brief case report might illustrate my point.

B.W., a forty-seven-year-old white male executive was referred for treatment of a depression. He had the classical symptoms of psychomotor inhibition, low point in the morning, crying spells, loss of interest, feelings of hopelessness, and occasional suicidal fantasies. In addition to these he had strong guilt feelings about having continued to masturbate after he was married at the age of twenty-seven. These guilt feelings were intensified by the fact that the patient's masturbatory fantasies had had a strong homosexual tinge.

A psychotherapist who had treated the patient for a year before we saw him had permitted the patient to spend many interview hours ventilating his guilt. The therapist then made the mistake of trying to interpret to the patient his homosexual fantasies. Latent homosexuality, mother identification, incest wishes, and a number of other psychonoxious mechanisms were illumed, and as time went on the patient got worse. He became more self-accusatory, he was convinced that he was a "queer" and he attempted suicide by cutting his wrist. This led to hospitalization and a change of doctors. The patient was given eight electroshock treatments, psychotherapy was carried out at the level of reassurance and ego strengthening, the patient was told that what he had done twenty years ago could not cause his present depression, the psychobiology of the hormonal system was explained to him, and after ten weeks of treatment the patient was discharged as having recovered.

I am certain that all psychiatrists have seen many cases similar to the one just described. Another point that comes to mind is that these patients respond much better to directive than to

nondirective therapy. We all have seen patients who, after having been subjected to non-directive therapy complain that "the doctor never *told* me anything." For the therapist to sit back passively and allow the patient to talk at random and for the therapist occasionally to utter a semi-directive interpretation which may be apprehended by the patient in any one of a number of ways could be hazardous. We all have seen patients in whom involutional depression has been treated non-directively and who have a feeling similar to that of a person who stands with both feet firmly planted in mid-air.

Devotees of the analytic school sometimes assert that, while many psychiatric conditions may respond to a non-analytic approach, whatever adjustment the patient makes is only temporary and there will be a recurrence. Clinical experience has taught that as far as the involutional depressions which make their first appearance in midlife are concerned, recurrences are relatively uncommon regardless of the type of psychotherapy employed. Let it be understoond that I am not opposed to analytic orientation *per se*. Having been trained in an analytic milieu, I am not unmindful of the value and benefits of analytic knowledge. In many psychiatric disorders other than involutional depressions the analytic approach and interpretation is the best therapy.

What I have just said applies to the true involutional melancholias as well as to the depressions of the manic-depressive type. We also include the so-called catathymic depressions—reactions in which there is a harping on one set depressive topic and not as much mood displacement as one finds in the so-called involutional melancholias. Even when we treat depressions with pronounced somatazation reactions, it sometimes is hazardous to get too deeply into interpretive therapy. However, it usually is quite safe to explain to the patients the effect of conditioning, the pathophysiology of the psychosomatic disorders, and to desensitize them on the matter of guilt feelings that they might be "putting on" or "imagining" their symptoms. In cases where somatazation reactions predominate, the fewer medications used the better. To fill a patient up with a variety of sedatives and the entire gamut of the newer wonder drugs merely tends to fix the patient's attention on the somatic aspects of his problem. By the same token, it is hazardous to delve too deeply into unconscious conflicts and to bring to the surface material that is psychonoxious. In depressions where paranoid thinking plays a prominent part, the psychotherapist must be extremely careful in interpreting the paranoid reactions to the patient. In most cases it is wiser and safer to stay away from any interpretation of latent homosexual wishes and their projections. Diethelm[6] states, "A thorough analysis for possible dynamic factors should be delayed until the depression has cleared up. Delusions of sin, especially in the sexual field, and guilt to actual past misdemeanors are viewed similarly. One should, however, always offer an opportunity for discussions which are terminated by constructive formulation." In other words, suppressive therapy should not be carried to the point where the psychotherapist completely ignores the patient's complaints. To tell the patient to forget it and that there is nothing wrong with him and then to prescribe a little more bromide is anything but good therapy.

If patients have religious scruples, it is usually well to have pastoral counseling. However, it must be remembered that the clergyman is not a healer. He is a helper and should work in cooperation with and under the supervision of the attending physician. While it is usually preferable that the assisting clergyman has some psychiatric orientation, I have found a number of clergymen who have an almost instinctive knack for helping people. The wise counselor will avoid too deep penetration into the philosophical aspects of the patient's religious preoccupations. He must be just as careful to guard against mobilizing guilt reactions as is the psychotherapist.

In some of the late involutional depressions, patients engage in deviations from the culturally acceptable patterns in the sexual sphere. It was formerly believed that these sex perversions in elderly persons were evidences of ethical deterioration on the basis of brain degeneration. However, we see many cases in which there is no evidence of deterioration (normal test results in the battery of psychometric examinations that we use as well as close clinical observation), and we feel that in cases where the patient has entered into biologically or socially forbidden channels, there has been a regression to infantile levels of gratification.[7] In the treatment of this particular aspect of the depressions it is safe to interpret to the patient the mechanism of regression. In fact, when the patient is able to realize what really is taking

(Continued on Page 458)

ACTH and Cortisone in Agranulocytosis

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THE very multiplicity of therapeutic agents used in agranulocytosis speaks for their lack of specificity and efficacy—pentnucleotide, folic acid, vitamin B_{12}, blood, BAL, et cetera.[4,10] This is so, irrespective of the cause—arsenicals, gold, dinitrophenol, aspirin, benzol, sulfa, aminopyrine, et cetera.[3,8,12,13] They do not affect the underlying pathological physiology, the maturation arrest of the precursors of granulocytes in the bone marrow. Administration of pentnucleotide may be followed by remarkable improvement, and immature granulocytes and monocytes may begin to appear in the peripheral blood with a rise in the total leukocyte count; however, the latent period between administration and evidence of improvement makes evaluation very difficult.[5] Indeed, a great many failures have been reported. Even instances of recovery may have been due to purely physiological factors rather than to the drug.

In recent years antibiotics have greatly aided in treatment by protecting the patient against infection during the period of recovery from sensitivity to the causative agents.[6,9,14] With the advent of ACTH and cortisone, which stimulate the bone marrow, it was natural that these hormones should be used in the usually self-limited sensitization phenomenon of agranulocytosis.[7]

A fifty-six-year-old woman was admitted to St. Andrew's Hospital on October 31, 1951, because of fever, sore throat, general malaise, prostration, nausea and vomiting. The present illness began during the first week in September when she noted a severe sore throat. She was hospitalized and treated elsewhere with large doses of penicillin and streptomycin, with no effect, from September 7 to 10. Her temperature reached 106° and she became semi comatose. On September 10 she received sulfonamides in the form of three pansulfa tablets every four hours. Each tablet contained 2.5 grains each of sulfamerazine, sulfadiazine and sulfacetamide. This medication was effective, and on September 14 she left the hospital. The white count was 6,500 on admission and 4,300 on discharge with a normal differential count on both occasions. Cultures from mouth and throat revealed mixed bacteria with no predominance. On September 21 she was seen again by her private physician, and while she still had a herpetic lesion on her lip, her condition was good. At that time her white blood count was 8,050, again with a normal differential. The patient had no further symptoms until one week prior to admission to this hospital when she ran a nail into her left palm. Following this there was a gradual recurrence and exacerbation of her original complaints. She denied taking any medication except Anacin and aspirin in large amounts (two to three tablets every two to three hours).

She had had stiffness and pain in her fingers and knees for many years, for which she took aspirin and Anacin. She had also tried a great many different "shots" with no benefit. She had received none since the present illness started. She had had acute hives in June, 1951, due to undetermined cause. The family history was not remarkable.

When first seen in consultation on November 1, 1951, the patient was moribund. Her temperature was 105°, pulse rate 135, and respirations 35. The mucous membranes of the mouth and throat were covered by a thick, grey membrane. She was unable to give the history. White count was 450 on admission, October 31, and 400 the next morning. All cells were lymphocytes. The sedimentation rate was 123 mm. in one hour, hemoglobin 11.3 gm. per 100 cc. (77 per cent) and red cell count 4,000,000. Blood pressure was 90/60. On the morning of the second hospital day she had already received 2,000,000 units of penicillin intravenously and 400,000 units every four hours intramuscularly, as well as streptomycin 0.5 gm. twice daily, without apparent effect.

Because death seemed imminent and no evaluation could be made as to the status of the adrenals, both ACTH and cortisone were started in spite of the well-known apparent effects of these agents on immunity and the possible antagonism of ACTH and cortisone.[1] That the immunity-reducing effect is only apparent is substantiated by the fact that the steroids actually increase effectiveness of treatment with antibiotics in conditions such as brucellosis. Further, a high corticoid level is not incompatible with high ACTH output in shock and stress.[2] Moreover, I believed the antibiotics would cover any decrease in antibody titre. ACTH was given in 25 mg. doses every six hours, and cortisone was given intramuscularly, 300 mg. the first day, 200 mg. per day on the second and third days, and 100 mg. daily thereafter. The dose of both ACTH and cortisone was gradually reduced, as was that of penicillin and streptomycin, as the dramatic response of the patient warranted (Table I and Fig. 1) and the blood picture dictated.

From the Allergy Clinic, Department of Internal Medicine, University of Minnesota.

TABLE I.

Date	Hemoglobin	Red Blood Cells	Leukocytes	Neutrophiles	Lymphocytes	Monocytes	Penicillin	Streptomycin	ACTH	Cortisone
10/31/51	77% (11.3)	4,000,000	450		100%		2,000,000 IV.	1 gm.		
							1,600,000			
11/1/51			400		100%		1,600,000	1 gm.	100 mg.	300 mg.
11/2/51			400	8	88%	4	1,600,000	1 gm.	100 mg.	200 mg.
11/3/51			2,550	34	64%	2	1,600,000	1 gm.	60 mg.	200 mg.
11/4/51			8,150	38	62%		1,600,000	1 gm.	60 mg.	100 mg.
11/5/51			12,550	40	60%		1,600,000	1 gm.	60 mg.	100 mg.
11/6/51			14,950	61	36%	3	1,600,000	1 gm.	40 mg.	
11/7/51			20,750	65	35%		1,600,000		40 mg.	
11/8/51			23,200	68	32%		1,200,000		40 mg.	
11/9/51			21,350	70	30%		800,000		20 mg.	
11/10/51			27,000	82	18%		400,000		10 mg.	
11/11/51			14,000	69	31%		400,000		10 mg.	
11/12/51			12,600	70	28%	2	400,000			
11/13/51			8,500	76	24%		400,000			
11/14/51			7,400	76	24%					
11/15/51			7,300	58	42%					
11/16/51			6,600	73	27%					

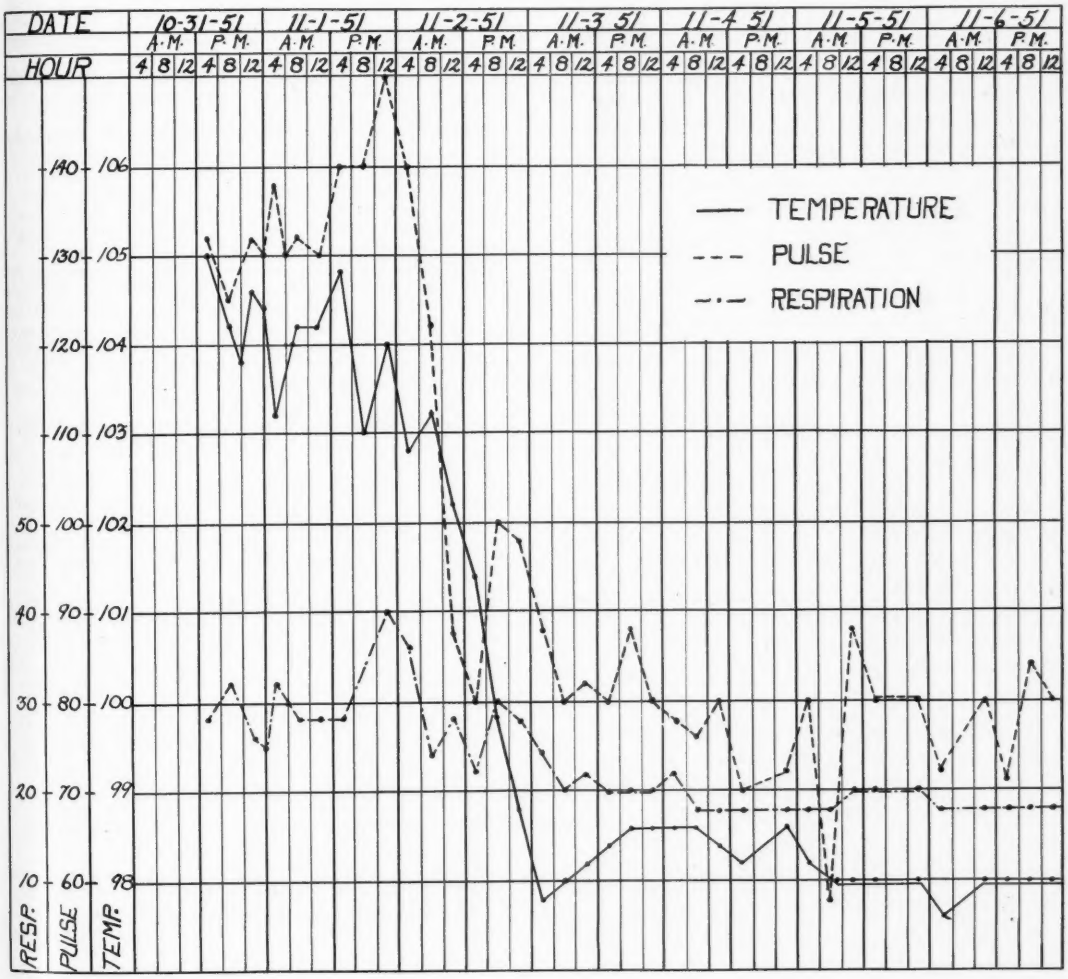

Fig. 1.

On November 8, 1951, sternal marrow and peripheral blood were examined by Dr. S. T. Nerenberg, who gave the following report:

"*Peripheral Blood.*—Examination of the red blood cells shows moderate anisocytosis with minimal poikilocytosis. Polychromasia is also minimal. The platelets are normal in number and appearance. Examination of the white blood cells shows the following differential: neutrophils, 80 per cent; lymphocytes, 17 per cent; monocytes, 1 per cent; eosinophils, 1 per cent; normoblasts, 1 per cent. There is a very marked shift to the left in the neutrophil series. Some of these cells appear to be blast forms.

"*Bone Marrow Aspiration.*—Bone marrow hematocrit shows the following percentages: fat, 2 per cent; ME layer, 14 per cent; plasma, 45 per cent; red blood cell layer, 39 per cent; total, 100 per cent. Examination of the bone marrow smears shows a marked increase in number of developing neutrophils. There is not an increase in the number of myeloblasts. The cells in the red blood cell series are decreased because of the marked increase in the neutrophilic series. Megakaryocytes are normal in number as are their developing platelets. Examination of the paraffin sections of the bone marrow shows a moderate hyperplasia of the marrow elements. A moderate amount of fat is still present.

"*Comment.*—The first peripheral smear seen on this patient was highly suggestive of myelogenous leukemia. However, subsequent smears and the bone marrow aspiration fairly definitely rule out leukemia.

"*Diagnosis.*—(1) Leukemoid reaction. (2) Neutrophilic leukocytosis. (3) Hyperplastic bone marrow."

On November 16, 1951, the patient was discharged with a normal blood picture and no acute symptoms.

Summary

A case of agranulocytosis is reported in which treatment of a moribund patient with ACTH and cortisone combined with penicillin and streptomycin was dramatically successful. A marked leukemoid reaction in the bone marrow was noted which abated with cessation of steroid therapy. The immediate response in this patient, confirming other reports in the literature since and before this patient was treated, would indicate that corticotropin and/or cortisone with antibiotics should be the treatment of choice in severe agranulocytosis.[11,15]

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(Continued from Page 446)

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Minnesota Automotive Crash Injury Research Program

A. B. ROSENFIELD, M.D.
Minneapolis, Minnesota

IN a further effort to decrease injuries and deaths on the highways of Minnesota, the Minnesota State Medical Association, the Minnesota Hospital Association, the Minnesota State Highway Patrol and the Minnesota Department of Health have undertaken a joint research program in collaboration with Cornell University Medical College. This study will begin about August 1, 1956, and will continue for two years. It will have as its primary purpose the gathering of factual information on the specific causes of injury to the occupants of passenger automobiles involved in accidents occurring on the rural highways of Minnesota. The Automotive Crash Injury Research project at Cornell University Medical College will integrate the Minnesota study into its interstate plan which is composed of the following other states engaged in similar research: Virginia, Maryland, North Carolina, Connecticut, Indiana, Arizona, Vermont, Texas, California, and the City of Minneapolis as an urban study.

The Cornell Automotive Crash Injury Research has been in progress since 1951 and is an outgrowth of similar Cornell studies in the aircraft field. It is sponsored by the Armed Forces Epidemiological Board under the Commission on Accidental Trauma, and the United States Public Health Service, with additional funds recently provided by the Ford Motor Company and the Chrysler Corporation.

The findings produced by this interstate project to date have identified the most frequent causes of injury in passenger automobile accidents as: ejection from doors which open as a result of impact on some section of the car other than the door itself; contact with the instrument panel; contact with windshield and header strip; contact with steering wheel and steering components; and contact against door structures. The information produced is used by automobile manufacturers as a basis for planning design changes in automobile structures aimed at reducing the frequency and severity of injuries in accidents.

The results can already be seen in many new automobile models now on the highways. Structural changes and new equipment include a new type door latch designed to keep doors closed under crash conditions, a steering wheel designed to absorb impact injury and to reduce injury to the chest, and optional installation of padding with a high energy absorbing characteristic and seat belts. These design improvements have been made possible only through the complete and thorough medical data contributed by physicians. The specific information on the nature, location and extent of injuries to automobile accident victims recorded by physicians is being correlated with information on the exact causes of injury submitted by state police and highway patrolmen investigating the accidents. Special photographs showing car damage, both inside and out, and all pertinent accident details are also submitted by the investigating officer.

Now that certain manufacturers are introducing design changes based on findings from the highways of the states participating in the study, Dr. John O. Moore, Director of the Cornell Automotive Crash Injury Research, points out that a new pressing problem has been created by the need to evaluate the precise degree of improvement which these engineering changes have had in controlling injuries when the new automobiles are involved in accidents.

The special medical form to be filled out by physicians is brief and the number of cases handled by each physician will be low. First of all, while the period of study will be two years it is subdivided into periods of six months each in selected sample areas. The study is limited to accidents investigated only by state highway patrolmen on rural highways in certain designated areas and for limited periods of time. No urban areas are included. Furthermore, the study is limited to

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passenger cars and is not concerned with occupants of buses, motorcycles or pedestrians.

Complete details of all injuries, no matter how minimal, with their degree and location are most important. This, however, will not be a formidable task since 75 per cent of persons injured in automobile accidents suffer only minor injuries. The request for the medical report will be made by the Minnesota Department of Health, usually through the state highway patrolman, and a special medical form will be supplied. No forms should otherwise be filled out. The completed medical form should be mailed promptly to the Minnesota Department of Health, University Campus, Minneapolis 14, who will be responsible for collecting the accident forms, photographs, and the medical forms.

The data submitted by physicians are for research purposes only. Under the laws of Minnesota such data, as well as data collected in the Minnesota Maternal Mortality Study, the Hennepin County Perinatal Mortality Study and other studies carried out by the Minnesota Department of Health in co-operation with other organizations and agencies, are confidential and can only be used for research purposes. Furthermore, the names of the physician and patient are recorded on a perforated section of the medical report which is removed and destroyed as soon as injury and accident facts are recorded on I B M punch cards.

Automobile manufacturers have already found the data of value in the improvement of safety features in automobile design. Insurance, the military, public health, safety and enforcement groups will also find the data useful in enforcement and educational efforts. In addition, these data are producing statistics which promise to be of value to physicians treating auto crash victims and the Traumatic Committee of the American College of Surgeons has expressed great interest in the study.

Prompt submission of reports on all automobile accident-injury patients will play an important part in the prevention of deaths or injuries resulting from auto accidents in the future.

HARMFUL EFFECTS OF INTERPRETIVE PSYCHOTHERAPY

(Continued from Page 453)

place, there frequently is alleviation of his guilt and also an improvement in his sexual behavior. However, when there is objective clinical evidence of beginning deterioration, interpretive psychotherapy is to no avail. Moreover, when there is deterioration, the prognosis for improvement in the individual's sexual behavior is and must remain doubtful. We usually advise that these patients be kept under fairly close observation and that they never be permitted to be with younger people unless accompanied by some responsible person.

Conclusion

There are two types of psychotherapy: expressive or deep; and repressive or supportive.

2. Whether the involutional depressions are exogenous or endogenous is debatable. However, there probably is no such thing as a purely endogenous or purely exogenous depression.

3. In certain of the involutional depressions expressive therapy with interpretation is hazardous and at times even harmful.

4. In certain of the involutional depressions suppressive therapy and management at the level of reassurance, together with careful attention to the patient's social adjustment, suffice.

5. The idea that unless the person with a depression is treated analytically, the illness will recur is erroneous.

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1044 Lowry Medical Arts Building

Medicatio Inhalatiorum

MAURICE S. SEGAL, M.D.
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INHALATION therapy has attained amazing prominence since the ancient and empirical practice of inhaling medicated vapors. The remarkable anatomical structure of the lungs, with its tremendous inner surface area and unique low pressure, low resistance and remarkable distensible circulatory apparatus, has made it possible to achieve high therapeutic levels at parenchymal sites with the use of therapeutic agents administered by the inhalatory and parenteral routes, alone or combined. To a growing list of directive terms must be added the "pulmonary route."

Inhalation therapy in its skilled practice covers a wide spectrum of physiologic and pharmacologic knowledge. The broad concept of the alveolar-capillary membrane includes all of the anatomic and physiologic road blocks in normals from the atmosphere to the tissue cells. The anatomic structures, to mention some, include the tracheobronchial tree, alveolar membranes, elastic and connective tissue fibrils, lymph channels and contents, capillary membrane and contents including the red blood cell envelope and hemoglobin, blood plasma and contents and ultimately the tissue spaces and their contents. In the sick patient further road blocks appear in the form of inflammatory products—cells, detris, fibrin, collagen, et cetera. The physiologic processes of ventilation, distribution, diffusion and perfusion influence and are influenced by the "alveolar-capillary membrane." Indeed, oxygen and carbon dioxide have a rough road, readily beset by many obstacles to interfere with the process of external and internal respiration.

There has been tremendous interest in the use of (1) therapeutic aerosols, (2) the hazards of oxygen therapy, and (3) pressure breathing therapy. Several of these advances will be discussed.

Therapeutic Aerosols

The clinical acceptance of the value of properly administered bronchodilator aerosols for the relief of bronchospasm has become widespread. We recently demonstrated in the laboratory considerable improvement in mechanical resistance, pressure-volume relationships, lung compliance and the actual work of breathing after the inhalation of bronchodilator aerosols. Many investigators have stressed the importance of obtaining high pulmonary parenchymal levels with aerosols of penicillin and streptomycin in resistive bronchopulmonary infections and in brochiectatic and atelectatic areas of lung. Others have stressed the useful antiphlogistic action of the corticosteroid aerosols in patients with allergic and inflammatory lesion in the upper respiratory passages; the value of detergent aerosols, enzyme aerosols and alcohol vapors as bronchial cathartics, muco-lytic and pus-lytic and anti-foaming agents respectively, and finally the appreciation of the greater usefulness of cold water vapor aerosols with continuous nebulization techniques (in the croupy disorders) as compared to the ancient steam kettle and the assortment of messy croup tents formerly employed.

Hazards of Oxygen Therapy

The emergence of oxygen as a double edged sword, although fully appreciated by many, has unfortunately been slow in universal recognition. Oxygen should be regarded as a drug with strict limitations and definite dosages (concentrations) —both useful and devastating. The physician must learn to prescribe the exact concentration needed, the duration of therapy and the specific type of apparatus best suited for the patient and his illness.[1,2] The tremendous increase in the incidence of retrolental fibroplasia has been attributed to the increased and casual use of high concentrations of oxygen without regard for duration of therapy and the need for gradual reduction in concentrations in the premature infant. It has also been attributed to the use of respiratory depressing drugs and hypoxia-inducing anesthesia in the mother during delivery. It would appear that any form of therapy which permits relative hypoxia by abrupt changes from high to low oxygen tensions in the premature infant may cause retrolental fibroplasia. Oxygen should be reserved for the premature infant with manifestations of asphyxia and then should be administered in concentrations

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not exceeding 40 per cent properly humidified and for as short a time as possible. One should always be certain of a clear tracheo-bronchial pathway.

The danger of respiratory acidosis is always present in patients with chronic hypoxia, secondary to chronic pulmonary emphysema or cardiopulmonary heart disease. This may occur spontaneously in the course of the disease or be induced by an infection of the respiratory tract or by the inadvertent administration (paradoxical therapeutics) of *high* concentrations of oxygen along with respiratory depressing drugs. The basic defective mechanism which is responsible for the progression of this syndrome is inadequate alveolar ventilation with marked retention of carbon dioxide. The carbon dioxide retention usually brings about a compensatory increase in the alkali reserve (metabolic alkalosis) and a fall in serum chlorides and increase in urine chlorides.[3] Failure in compensatory rise in buffer base and an ultimate drop in arterial pH may develop rapidly, leading to a fatal issue.

If respiratory acidosis develops during the inadvertent administration of high concentrations of oxygen, the method should be changed to the nasal catheter technique, employing oxygen flows of 1 liter per minute, with gradual daily increases.[1,2] If an opiate or demerol has been the cause of respiratory depression, it should be countereacted at once by the administration of Nalline.[1]

Pressure Breathing Therapy

A very interesting chapter in mechanical respiration has been the recent advances in pressure breathing therapy.[4,6,7,8] Many investigators have observed the value of intermittent positive pressure breathing during inspiration with the simultaneous administration of bronchodilator aerosols, noting relief of bronchospasm, improvement in ventilation and distribution factors, and elimination of carbon dioxide. In the laboratory we have observed a decrease in mechanical resistance, improvement in lung compliance and also a progressive rise in venous pressure after fifteen minutes of this form of therapy.[5,6] Because of inadequate circulatory dynamics in patients under circulatory stress this form of pressure breathing should not be employed in patients who are on the verge of or in peripheral vascular collapse, heart failure or those having spinal anesthesia. The venous-gradient return to the right side of the heart may become more inadequate and a further drop in cardiac output may ensue.[7]

More recently another form of pressure breathing has been described, wherein alternating positive and negative pressures are applied to the oro-pharynx.[6,8] Following the positive pressure during inspiration, a very rapid changeover to high negative pressures during expiration is achieved ("exsufflation with negative pressure").[8] This is associated with high peak flow rates during expiration approaching those noted in a successful cough. It has been shown that with positive and negative pressures the mean mask pressure approaches more closely that of ambient air[6] and is furthermore safer in reconstituting the venous gradient return to the right side of the heart.[7]

In the laboratory we have noted in patients with chronic pulmonary emphysema actual bronchial chattering in our tidal volume curves and pneumotachograph tracings during the high expiratory flow rates.[5,6] This is a form of ball valving described by others in these patients during coughing and vigorous expiration. In patients with chronic pulmonary emphysema the critical velocity rate may be below 500 cc./second. Above this rate laminar flow is converted to turbulent flow. Added pressures will probably not improve flow rates under such circumstances in patients with chronic pulmonary emphysema.[9] This form of therapy has been found most useful, however, and even life saving in patients with ineffective cough mechanisms and trapped secretions secondary to bulbar poliomylitis, atelectasis, bronchiectasis, lung abscess and inhalation poisonings.[6,8]

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[1]Albertson, H.A. and Trout, H. H., Jr.: *Antibiotics Annual* 1954-55, Medical Encyclopedia, Inc., New York, N.Y., 1955, pp. 599-602.

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Congenital Hemihypertrophy and Hemihypoplasia

DORRANCE BOWERS, M.D.
Rochester, Minnesota

CONGENITAL hemihypertrophy and hemihypoplasia are anomalies that provide grotesque caricatures of the minor asymmetries present in every human body. The purpose of this communication is to present photographs of patients with each of these rare anomalies.* These photographs call attention to the cosmetic and tailoring problems and the difficulties with gait and posture that such patients face. Physicians may fail to realize the distress that malocclusion of the teeth[1] may cause these unfortunate victims of nature's caprice.

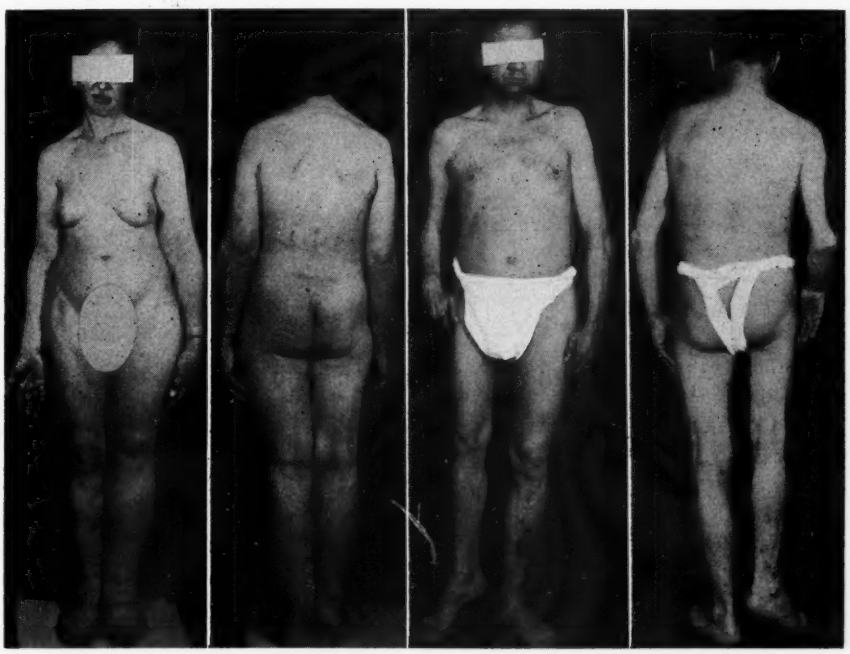

Fig. 1. Congenital left hemihypertrophy. Note the asymmetry of the mouth, trunk and extremities. The right tibia was 37.5 cm. in length, whereas the left was 40 cm., as measured from roentgenograms. A hemangioma at the left angle of the mouth and another on the left forearm are not well shown.

Fig. 2. Congenital right hemihypoplasia. Again, note the asymmetry of the mouth, trunk and extremities. An associated congenital anomaly of the right elbow also is shown.

The right half of the body of each of these two patients has been smaller than the left since birth (presumably also since before birth). The left half of the body may have become larger than the right because of excessive development of the left side, referred to as hemihypertrophy (Fig. 1). The discrepancy also may arise because of incomplete development of the right side; this particular condition has been referred to as hemiatrophy but it is designated more correctly as hemihypoplasia (Fig. 2).

The physician may recognize readily distortion resulting from the unequal size of the two halves of the body in patients who have such anomalies. However, he may be unable to specify which is

(Continued on Page 466)

*The author is indebted to Dr. L. E. Ranta, Assistant Director-Medical, Vancouver General Hospital, Vancouver, British Columbia, for permission to publish these photographs.

Case Presentations

Bronchogenic Carcinoma with Unusual Presenting Symptoms

RICHARD F. KLINE, M.D.
Minneapolis, Minnesota

THIS is the history of a man with bronchogenic carcinoma, who, in addition to the symptoms associated with a pulmonary neoplasm, complained of pain in the hip; who, following examination, was found to have clubbing of the fingers and auricular fibrillation.

This fifty-six-year-old white man was admitted to the Abbott Hospital, Minneapolis, Minnesota, on October 30, 1953. During the preceding two weeks he had had a productive cough, shortness of breath, loss of appetite, and recurrent febrile episodes. He had lost 10 pounds in weight. Several weeks earlier he had noticed some enlargement of his finger tips, and more recently he had had a persistent, aching pain in the right hip. He felt that he had not actually "been well since July or August." He smoked one to two packages of cigarettes daily.

The pertinent physical findings revealed a well-developed, white man with a blood pressure of 130/90. The radial pulse was irregular and difficult to obtain. The apical pulse rate was approximately 140. Thyroid, breast, and lymphatics were normal. The lungs were clear. The heart was moderately enlarged to percussion. There was a discrete area of tenderness over the crest of the right ilium. Examination of the genitalia, rectum, and prostate failed to reveal any abnormalities. There was moderate clubbing of the fingers.

The initial chest x-ray was reported as showing a slight thickening of the pleura at the base of the left lung with a rounded nodule in the base of the right lung (Fig. 1). This *probably* represents a childhood tuberculosis, the report stated, but a repeat x-ray film in six months should be taken to establish this diagnosis. Stereoscopic antero-posterior pelvic x-rays showed hypertrophic changes of the lower lumbar vertebrae. The hemoglobin was 14.1 grams; red blood cells 4.7 million; white blood cells, 13,800 with 75 per cent polymorphonuclear cells and 25 per cent lymphocytes; the sedimentation velocity was 37 mm. in one hour, and the serology was negative. The urinalysis and concentration tests were normal. The liver function tests were normal with the exception of a 15 per cent bromsulfophthalein retention at forty-five minutes.

Treatment consisted of an attempt to digitalize the patient. Following consultation the digitalis was stopped and quinidine was used. This produced a normal rhythm. His oral sepsis and pulmonary disease was

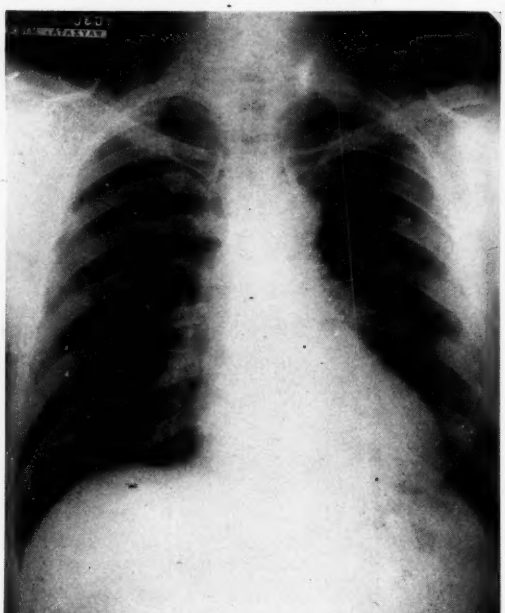

Fig. 1. Chest x-ray, October 30, 1953. In retrospect, the lesion appearing at the base of the right lung may have been neoplastic in origin.

treated with penicillin. He was discharged November 7, 1953, with relief of his pulmonary symptoms. The pain over the ilium persisted without explanation.

The auricular fibrillation recurred despite the quinidine. He was readmitted to Abbott Hospital on December 1, 1953. There had been little increase in his pulmonary symptoms; however, the clubbing of the fingers had increased and the pain in the hip was worse. Digitalis finally controlled the tachycardia. X-rays of the pelvis were again normal. Laboratory studies were normal except for the sedimentation velocity, which had increased to 76 mm. in one hour.

From the files of Drs. W. W. Rieke and R. F. Kline, Wayzata, Minnesota.

Dr. Kline is now associated with the Veterans Administration Hospital, Minneapolis.

The basal metabolic rate was normal. The patient soon developed a cough which was productive of small amounts of bloody sputum. Rhonchi were heard posteriorly in the left base. Sputum studies were negative for acidfast bacilli and tumor cells. A chest x-ray reported pleural thickening at the left costophrenic angle. Bronchoscopic examination was deferred because of the cardiac condition. Twelve days after this second admission a mass appeared over the crest of the right ilium. A biopsy specimen of this mass revealed a highly anaplastic metastatic adenocarcinoma (Figs. 2 and 3). Further x-rays and laboratory studies failed to reveal the primary lesion. It was presumed that the primary lesion was either in the lungs or the pancreas. The patient returned home on December 19, 1953.

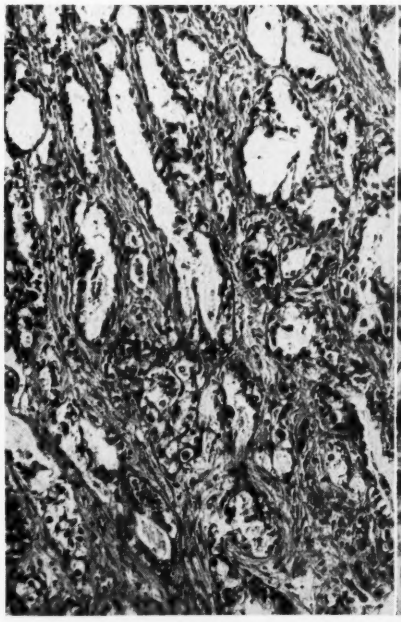

Fig. 2. Photomicrographs of biopsy from soft tissue along iliac crest. ×100.

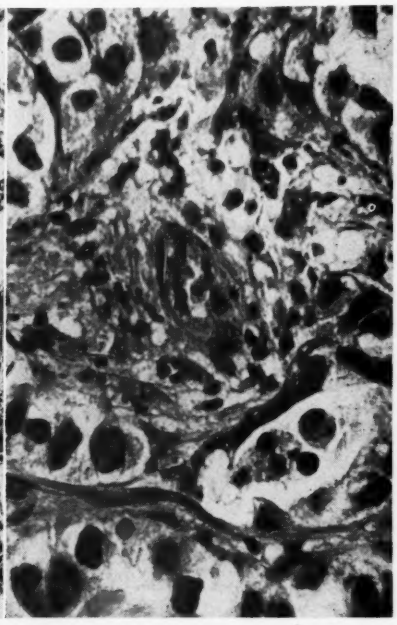

Fig. 3. Photomicrographs of biopsy from soft tissue along iliac crest. ×440.

He was admitted to Northwestern Hospital on December 31, 1953, for palliative x-ray therapy to the region of the right hip. While hospitalized, he developed an atelectasis of the left lower lobe of the lung, and he had pain and tenderness over the left fifth rib anteriorly. X-ray examination now revealed a mass in the left lower hilum (Fig. 4). He received palliative x-ray therapy to the lungs and mediastinum. There was further weight loss with increasing difficulty in controlling the heart rate with digitalis.

The patient was discharged from the hospital on January 22, 1954, and he died at his home on February 10, 1954.

Summary of Autopsy Findings

An ulcerating tumor measuring approximately 7 by 5 by 5 cm. was found along the right main bronchus about 1 cm. from the carina. The tumor extended into the mediastinum and into the left atrium of the heart at the point of entry of the superior pulmonary vein. A tumor nodule, presumably metastatic, 4 cm. in diameter, was found in the *right* lower lobe. The left pleural cavity was bound down by firm, fibrous adhesions.

Metastases (in addition to the one examined along the right iliac crest) were found in the liver, spleen, kidneys, ilium, and the adrenals, with the latter organs almost entirely replaced with tumor tissue.

"Sections of the primary tumor and the metastases showed it to be composed of aberrantly growing and infiltrating gland structures. In places the epithelium is in the form of tall columnar epithelium, with evidence of mucin secretion. In other areas the tumor is undifferentiated, markedly pleomorphic, and shows numerous mitotic figures."

Discussion

The presenting symptomatology of bronchogenic carcinoma, as illustrated in the above case, is often varied and baffling. This disease is notorious for first presenting symptoms and findings suggestive of a bronchopneumonia, responding temporarily to antibiotics. In addition to the usually described symptoms of cough, hemoptysis, chest discomfort, dyspnea, et cetera, one may be confronted with secondary phenomena which may or may not be metastatic. Central nervous system metastases are common for

bronchogenic carcinoma is second only to primary brain tumors in central nervous system malignancies. The very nature and location of this tumor readily permits widespread dissemination

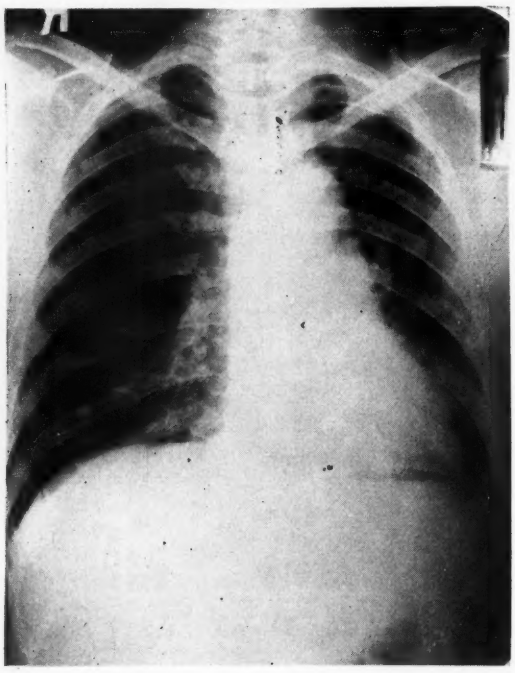

Fig. 4. Chest x-ray, January 6, 1954, first showing extensive changes in the left hilum.

of metastases. The chest x-ray may possibly reveal nothing or leave one with rather inconclusive findings. A knowledge of these secondary symptoms and findings will raise the physicians's index of suspicion and lead to early chest x-rays; and, even where these are negative, to cytologic, bronchoscopic, bronchographic, and planigraphic studies.

The skin and soft tissue are unusual sites of dissemination of bronchogenic carcinoma. Entieknap, noting the location of metastasis, reported an incidence of 2 per cent to skin or soft tissue.[3] In the above patient the soft tissue tumor was interesting. By so closely mimicking a bone lesion, it proved to be an enigma to all who examined the patient. Ultimately, it definitely established the diagnosis of malignancy.

Cardiac neoplams, although uncommon, are not as uncommon as generally believed; and the majority are metastatic in nature.[1,2] The usual site of the primary tumor is either the bronchus or the breast, with the former site predominating. Entieknap, in his series of 1,000 cases of cancer with reference to metastases, reported forty-nine metastases to the myocardium or pericardium, thirty-five of which had a primary site in the bronchus.[3] Ritchie reported a lesser, but high ratio implicating bronchial neoplasms in cardiac metastases.[4] Knowledge of a metastasis to the heart is usually found only at autopsy—physical findings or symptoms with reference to the heart being very uncommon. The metastatic spread to the heart in the above case would readily explain the refractoriness of the auricular fibrillation to the usual cardiac medications. White states that, having excluded all other possibilities of cardiac dysfunction, the rare possibility of a primary or secondary cardiac neoplasm should be considered.[5]

At present, the finding of distant metastases, in most instances, may be of little more than diagnostic help and may offer little definitive therapeutic benefit to the patient. Clubbing of the fingers alone or in association with long bone proliferation and arthropathies is a physiological phenomenon which may occur early in the course of the disease.[6,7,8] This phenomenon, known as pulmonary hypertrophic osteoarthropathy, was first described by Marie in 1890. It is characterized pathologically by a general increase in fibroelastic and connective tissue in the enlarged digits; the long bones show periosteal new bone formation. By virtue of the large number of theories offered to explain it, the etiology of pulmonary hypertrophic osteoarthropathy is obscure.[9-12,14] Reported incidences vary from 2 to 10 per cent of all bronchogenic carcinomas.[13,14,15]

One point should be emphasized: pulmonary hypertrophic osteoarthropathy often occurs early —while the pulmonary lesion may still be in a resectable stage. Pattison recently reported six cases of bronchogenic carcinoma with pulmonary osteoarthropathy, in five of which this was the earliest significant finding.[16] It is apparent then, that in some cases, we have available a diagnostic sign which may lead to the early diagnosis of a bronchogenic carcinoma.

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Traumatic Pancreatitis with Reference to the Abetting Influence of Pancreatic Stimuli

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THE occurrence of pancreatitis as a primary entity or as a complication is a serious one. The morbidty and the mortality of this condition is significant, the treatment is not specific, and the sequelae may be prolonged. Moreover, the diagnosis may be difficult, and often is one of elimination. There is almost no other organ in the body that is able to disguise its disease so well.

The diagnosis of acute traumatic pancreatitis in the absence of a penerating, or even a direct wound to the abdomen, is even a more difficult situation. In such cases, there are often more obvious injuries to call attention to other areas of the body, and deep lying damage thus remains unsuspected. The following is such a case.

Case Report

Three days prior to his University Hospital admission, the patient, a seventy-one-year-old white man, was the driver of one of the cars involved in a head-on automobile accident in which he suffered abrasions and lacerations to the face, scalp, and contusions to the upper abdomen, and chest. Following emergency treatment he was discharged to his home the evening of the accident in apparently good condition considering the magnitude of the damage to his car.

The next day he developed shortness of breath, auricular fibrillation and abdominal distention. He was hospitalized in his home area, and twenty-four hours later transferred to the University Hospitals. Roentgenological examination of the chest showed left ventricular enlargement and extensive pulmonary edema associated with pleural effusion and areas of linear atelectasis. The electrocardiogram showed left axis deviation, rapid auricular fibrillation with bursts of paroxysmal ventricular tachycardia of short duration together with changes suggestive of ischemia. Abdominal x-rays disclosed a picture compatible with severe ileus.

The past history was not remarkable except for a protracted history of alcoholism. A blood alcohol drawn at the time of the automobile accident revealed a level compatible with an extreme degree of intoxication.

Physical examination upon hospital admission revealed a well developed and well nourished seventy-one-year-old white man, in acute and severe respiratory distress associated with severe back and chest pain. The blood pressure was 130/70 mm. Hg, pulse rate was 144 per minute and his temperature was 102 degrees. The skin and nail beds were cyanotic. There were râles and ronchi in both lung fields. The heart was enlarged to the anterior axillary line, was grossly irregular, but with no murmur. The abdomen was very distended and without bowel sounds. There was direct tenderness in the epigastrium with mild rebound tenderness. The remainder of the physical examination was non-contributory.

Laboratory examination revealed a hemoglobin of 14.2 grams per 100 cc. of blood, white blood count was 6,300 with a normal differential count. Urinalysis showed a specific gravity of 1.018, albumen was 1 plus, and only occasional red and white blood cells were noted. Blood chemical examination revealed a BUN of 60 mg. per cent, creatinine of 1.8 per cent, carbon dioxide of 22 mEq./l., chloride of 112 mEq./l., sodium of 142 mEq./l., and potassium of 3.8 mEq/l.

The patient was obviously gravely ill and was thought to have traumatic myocarditis or infarct precipitated by the trauma of the steering wheel striking the chest superimposed upon arteriosclerotic heart disease. The etiology of the ileus was obscure but was considered to be on a reflex basis from the thoracic wall injuries. He was treated with rapid, digitalization and diuretics for the cardiac manifestations, and nasogastric suction to relieve his ileus. Intravenous fluids were necessary but the quantity was limited for the first twenty-four hours to prevent further pulmonary edema. Within twelve hours the heart rhythm had become regular and the pulmonary edema was relieved. The pulse rate continued between 100 and 140 per minute.

During the first four days in the hospital, the patient continued to be markedly confused and disoriented, with occasional episodes of gross tremor. This state was thought to represent a mild form of delerium tremens due to the history of chronic alcoholism.

Despite the prompt and dramatic improvement in this patient's cardiopulmonary status the severe ileus persisted out of all proportion to his other injuries. Exploratory laparotomy was considered at the time of admission, but was discouraged by his acute cardiac decompensation with pulmonary edema and the absence of any definite evidence of a perforated viscus. By the fourth day the reflex etiology for the persistent ileus seemed less tenable, and a serum amylase was drawn and found to be 386 units (normal 50 to 150 units). Continuous intestinal decompression and antibiotic therapy were continued and by the seventh hospital day (ten

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days after the accident) his ileus began to resolve. At this time the serum amylase had fallen to 143 units. He was discharged feeling well on the fourteenth hospital day.

Discussion

The problem of pancreatic injury was recognized as early as 1827 by Travers.[1] Naffziger and McCorkle[2] have emphasized the value of the serum amylase test in the recognition of acute traumatic pancreatitis. Interestingly, the experimental studies of Popper and Nechels[3,4] have shown that the resistance of the pancreas to injury by direct trauma is very great. In their animal studies, these authors were able to inflict considerable trauma to the pancreas and even open pancreatic ducts without finding fat necrosis or other evidence of pancreatitis when they sacrificed their animals. Significantly, they did not find this same to be true when only minimal trauma to the pancreas was inflicted after a pancreatic stimulus such as cholinergic drugs or food had been given. In such experiments even mild trauma resulted in a severe pancreatitis. From these studies, they have concluded that it is not the extensive pancreatic trauma that is of the greatest hazard, but rather trauma, even mild, associated with a pancreatic stimulus. Extensive pancreatic damage in the fasting state resulted in cessation of pancreatic secretion for a long enough period of time to allow the rents in the gland ductal system to be sealed by omentum or loops of bowel. However, with mild trauma combined with a pancreatic stimulus there resulted a flow of pancreatic secretions from the injured sites thus preventing sealing of the ductal system and with resultant pancreatic extravasation and fat necrosis. These experimental studies help explain the clinical observations of Warren,[5] who noted that the trauma to the abdomen in some patients with traumatic pancreatitis was trivial.

Likewise, this patient demonstrates the clinical application of this study. His accident occurred after an evening of dining and imbibing of alcoholic beverages which "set the stage" for his accident as well as his pancreatitis. His pancreatitis was not diagnosed until his persistent ileus seven days following the accident caused a search for other etiologic factors and a serum amylase was drawn.

Summary

A case of traumatic pancreatitis following an automobile accident associated with alcoholic ingestion is presented. The importance of food or alcohol as pancreatic stimuli in abetting pancreatic susceptibility to trauma is described. The diagnosis of acute pancreatitis under these conditions is easily overlooked if the possibility is not kept in mind. The problem is further complicated, as the signs and symptoms of pancreatic injury do not develop until some hours later, often ten to thirty-six hours after the initial trauma. The trauma to the abdomen may often be considered trivial.

It is suggested that it should be a common emergency room procedure to take this condition into diagnostic consideration when unexplained abdominal or back pain, fever, and ileus accompanies other traumatic injuries.

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HEMIHYPERTROPHY AND HEMIHYPOPLASIA

(Continued from Page 461)

the abnormal half, and he may be unable to determine whether the distortion results from hemihypertrophy or hemihypoplasia. While such ignorance may be embarrassing, it is minor, indeed, compared to the abysmal ignorance that surrounds the etiologic factors[2] concerned in these anomalies.

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Seminar

THE PYRROL PIGMENTS AND HEMOGLOBIN CATABOLISM

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(Concluded from June Issue)

Representation of Hemoglobin Catabolism by Pyrrol Pigment Excretion

On the basis of well-established structural relationships one gram of hemoglobin can give rise to approximately 34.7 milligrams of bilirubin and 35.1 mg. of mesobilirubinogen. Thus 100 gm. of hemoglobin yields 0.34 gm. iron representing four molecules of heme (ferrous protoporphyrin). The heme molecule ($C_{34}H_{34}N_4O_4Fe$) weighs 618, protoporphyrin 562, bilirubin 584 and mesobilirubinogen 592. 1 gm. of hemoglobin theoretically provides 37.5 mg. heme, 33.4 mg. protoporphyrin, 34.7 mg. bilirubin and 35.1 mg. mesobilirubinogen. A slightly larger amount of stercobilinogen, and slightly smaller amounts of d-urobilinogen or dihydromesobilirubin are provided. From the standpoint of urobilinogen group excretion it is believed best to employ the figure for mesobilirubinogen. On the basis of the figure just given, 100 mg. of mesobilirubinogen is equivalent to 2.85 gm. hemoglobin. The figure of 2.39, given nearly twenty years ago[26] and based on Eppinger's data,[22] is thus seen to be too low. Taking this correction into account, the normal value for W, the apparent hemoglobin wastage represented by the daily fecal urobilinogen in relation to total circulating hemoglobin,[26] is proportionately increased to a normal range of 0.5 to 0.7 per cent per day. While recognizing that this figure is probably still too low, it is true that use of the value W is often quite helpful in the more exact evaluation of urobilinogen excretion as related to the problem of anemia.

The first E. T. Bell Lecture of the Minnesota Pathological Society.

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This lecture has appeared in three installments, of which this is the third.

If it were clear that all of the protoporphyrin in the hemoglobin molecule were converted to bilirubin, that all of the bilirubin were excreted in the bile and thence into the intestine to be reduced in toto to the urobilinogen group and that all of the urobilinogen group was then excreted in the feces, the situation would be relatively simple and one could make an exact calculation of the rate of hemoglobin catabolism. The problem, however, is by no means as simple as this. There is first of all the possibility to be reckoned with that some fraction of the protoporphyrin is at once split into smaller fragments than bilirubin so that it would not be represented in the bilirubin of the bile or the urobilinogen of the feces. Bingold[93] has been the chief proponent of a theory that a certain fraction of the circulating hemoglobin is destroyed by the kidney. According to this concept, an unknown proportion of the protoporphyrin is split into two dipyrrylmethenes which then appear in the urine as colorless chromogens and may be detected by the "pentdyopent" reaction. This simply refers to a pink color reaction with an absorption band at 525 mμ observed after alkalinization and heating of the urine and addition of sodium hydrosulfite. Bingold demonstrated that the same reaction is observed if hemin is oxidized with hydrogen peroxide, then treated with sodium hydrosulfite. This is also true of human blood if first heated to 72° C. Bingold believed that the heat destroyed the catalase of the red cells thus exposing the hemoglobin to the oxidative activity of hydrogen peroxide. Goose blood, in which there is but little or no catalase, is decolorized by peroxide without preliminary heating. According to Bingold this is also true of the urine from human cases of hemoglobinuria, explained as due to a removal of catalase by the kidney.

Pentdyopent is also formed from bilirubin and urobilin (i- or d- but not l-), and indeed it seems

likely that its presence in the urine is more often associated with degradation of one of these substances. Nevertheless it has been observed in the absence of either bilirubin or urobilinogen, and Bingold believed that he was able to demonstrate it in the circulating red blood cells. He thought, however, that this might be related to anabolism rather than catabolism. There is no further information on this point. Although Stich has mentioned methods of quantitative determination of pentdyopent, exact techniques have not been described, and our own attempts† to estimate the substance in the urine have met with failure due to the capricious nature of the reaction itself, the lability of the substance and the many variables which influence the intensity of the color development. The color reaction in the urine is seldom intense enough to suggest that any important fraction of the destroyed hemoglobin for the period of time involved was represented. While the pentdyopent reaction undoubtedly indicates a catabolism to smaller molecules than urobilinogen, it is not believed to have appreciable significance from a quantitative standpoint. Nevertheless its presence might conceivably indicate that some larger fraction of the porphyrin or bile pigment moiety is being converted to other dipyrrylmethenes or smaller molecules not revealed by the pentdyopent reaction. Thus, mesobilifuscin, to be discussed in the following, is a naturally occurring dipyrrylmethene which does not give the pentdyopent reaction.

Except for the possibility that a small fraction of the destroyed hemoglobin is at once converted to a dipyrrylmethene such as represented by pentdyopent, rather than to bilirubin, there is no available evidence that the bilirubin excreted in the bile does not represent hemoglobin catabolism in nearly quantitative fashion. No reliable evidence has been brought forward that any fraction of the bilirubin formed in the body is destroyed and reutilized. In the presence of regurgitation jaundice, of course, variable amounts of bilirubin are excreted in the urine, and in any patient with jaundice some portion of the bilirubin being formed remains for variable periods in the blood and tissues. Except for these abnormal situations, however, there is every reason to believe that bilirubin is efficiently excreted by the liver into the bile. As already mentioned, we have only fragmentary information about the amount of bilirubin in the twenty-four-hour bile of human beings. There are many observations on the bilirubin content of gall bladder bile, fistula bile in the presence of a T tube, and duodenal contents under varying circumstances, but these are obviously of limited value. The available data have recently been reviewed by With.[23] So far as can be determined, only the observations of Eppinger[22] are reasonably acceptable on the basis of relating to the whole bile in instances of total biliary fistula in relatively normal human beings. Eppinger[22] found 300 and 370 mg. per twenty-four hours in these two individuals. The amount of the total circulating hemoglobin was not determined. If one assumes that it was 750 gm. (blood volume of 5 liters with 15 gm. of hemoglobin per 100 cc.) a red cell life span of approximately seventy-seven days may be calculated. This is shorter than the value of 120 days which in recent years has been determined by various investigators employing tagging with N_{15}, Cr_{51}, Fe_{59}, sulfhemoglobin or immunological methods of the Ashby type. Nevertheless, Evans has recently postulated an average life span of 90 to 100 days on the basis of two populations of red cells, a very short lived group living four to five days and a larger group living in the neighborhood of 120 days.[95] Evans' calculations were based on several unproven assumptions which may or may not be valid. Evans also believes that earlier data of Finch and associates,[96] when employed in his calculation, provide further evidence of shorter lived red cells in normal individuals. Finch's data were obtained by means of intravenous administration of Fe_{59}.

Although Eppinger's values for bilirubin were based on a spectrophotometric method, it is possible, as the bilirubin absorption is rather nonspecific, that other substances were included and that his values were somewhat too high. It is evident that studies with modern techniques in cases of total biliary fistula, otherwise in relatively good health, are very much needed. Such patients have become extremely rare as nowadays the establishment of a complete external biliary fistula is generally regarded as undesirable even in a patient with inoperable carcinomatous biliary obstruction (it is obvious that for the purpose of measuring the total bilirubin excretion a complete billiary obstruction would be essential).

For a normal individual such as just referred to, having a total circulating hemoglobin of 750

†With S. Schwartz, unpublished.

grams, a red cell life span of 100 days would account for 260 mg. per day of bilirubin in the bile, if the foregoing assumptions are valid. For a life span of 120 days about 216 mg. per day would be provided. As will be noted in a moment, there is recent evidence which has been thought to indicate that not all of the bilirubin in the bile or the resultant urobilinogen of the feces is derived from destruction of the hemoglobin in mature circulating red blood cells. The published data for the daily urobilinogen excretion[97] in the feces have consistently given values lower than those just discussed for the bilirubin of the bile. While the question of bilirubin reabsorption from the intestine cannot be answered with finality, the majority of evidence indicates that it is negligible if it occurs at all. There is general agreement that the urobilinogen in the feces in twenty-four hours in significantly lower than the amount of bilirubin in the bile. If Eppinger's figure of 300 and 370 mg. is accepted, it is clear that the deficit would be considerable. In 1936[97] the author reported an average of 180 mg. per day for seventeen normals. Of these, however, but two were females. Furthermore, the method employed at that time was not as precise as that which has been used since 1944.[98] Heilmeier and Oetzel[99] had previously reported 146 mg. per day as the average value in fourteen normals of both sexes. Quite recently Dr. Sigrid Gilbertsen in this laboratory has obtained a mean value for both sexes of 140 mg. per day. It is seen in any event that even if one accepts a figure of 210 to 260 mg. per day for the bilirubin of the bile, a figure which, as just seen, would correspond with a life span of the red cells of 100 to 120 days, there is a significant deficit between bilirubin and urobilinogen to be accounted for. Several possibilities require consideration:

1. Incomplete reduction of bilirubin to the urobilinogen group. It has already been noted that even normal feces contain significant amounts of bilirubin. This in itself suggests that intermediaries, such as mesobilirubin and dihydromesobilirubin might also be present. Both of these have indeed been identified in feces containing mainly d-urobilinogen,[77] but, as already mentioned, it appears quite likely that the latter may be a normal intermediary compound on the pathway to stercobilinogen, also that it may be isomeric with dihydromesobilirubin. It should be noted that the latter substance also exhibits an Ehrlich reaction so that it would contribute to the colorimetry of urobilinogen based on this method, yet no information is available as to comparative optical density of its aldehyde compound. It is believed quite unlikely that the contribution of dihydromesobilirubin to the total Ehrlich reaction for urobilinogen, is very large, though, obviously, further studies of this question are needed. It appears that dihydromesobilirubin does not form a urobilin; at least when subjected to the iodine conversion method in petroleum ether,[80] only mesobiliviolin has been observed.** W. C. Meyer[86] has suggested, however, that dihydromesobilirubin may be the precursor of a third urobilin which he has observed, one characterized by a copper reaction differing from that of urobilin or stercobilin. No further information about this compound is thus far available.

2. Reabsorption of members of the urobilinogen group with chemical alteration or reutilization. As already mentioned, there is strong evidence of an enterohepatic circulation of urobilinogen but no evidence that it is reutilized in heme synthesis. In fact, London, employing N_{15} tagging, was unable to find any evidence of reutilization.[100] If one assumes that from 50 to 100 mg. of the total urobilinogen group are reabsorbed from the colon and returned to the liver each day in a normal adult, one would expect that if there were a complete enterohepatic circulation, a corresponding concentration in the normal bile would be found. The observations of Jankelson[101] and Sborov,[102] together with many older observations as reviewed in considerable detail by With,[23] indicate that the amount of urobilinogen group substances in the bile is relatively quite small, probably not more than 1 per cent of the fecal urobilinogen. Thus if the discrepancy between bile bilirubin and fecal urobilinogen is due to partial reabsorption of urobilinogen, it follows that this fraction must undergo alteration or destruction in the liver or elsewhere in the body so that it is no longer detectable as urobilinogen. In this connection it is of some importance that liver homogenate was shown by Felix and Moebus[103] to cause a rapid disappearance of added mesobilirubinogen. Another possibility which would explain the discrepancy is that some fraction of the bilirubin or urobilinogen group derived from it is split into smaller mole-

**Unpublished observation with P. T. Lowry.

cules. This is a very important question, and I will attempt to deal with it in a moment in relation to the problem of bilifuscin and mesobilifuscin.

First, however, it is desirable to give some attention to the interesting question as to whether all of the fecal urobilinogen is derived from the destroyed hemoglobin of circulating red blood cells. In the earlier literature one finds this assumed by Eppinger and Charnas,[104] but strongly doubted by George Whipple,[105] who first conceived the idea of a "pigment pool" from which unused pigment such as bilirubin or urobilin might be excreted without having been in the hemoglobin molecule. Whipple[105] and Castle and Minot[106] both favored the idea that in pernicious anemia there is a faulty utilization of pigment because of the maturation arrest in the bone marrow, with resulting accumulation and excessive excretion. Our own data in pernicious anemia, obtained with Jones,[107] supported this view. In this latter study it was observed that in pernicious anemia in relapse the hemoglobin level often remained relatively static despite marked excesses of urobilinogen in the feces and at the same time without any increase of the reticulocyte percentage, indeed with marked reduction of the absolute reticulocyte count. In other words, a striking imbalance between the apparent rates of destruction and regeneration, which by simple calculation ought to have produced a much more rapid decline of the hemoglobin level, nevertheless failed to do so. It was concluded on this basis that a considerable fraction of the fecal urobilinogen in pernicious anemia in relapse was derived from sources other than the hemoglobin of destroyed circulating erythrocytes.[26] Prior to this, Jedlicka[108] suggested that the excessive pigment excretion in pernicious anemia might be due to fabrication and destruction of unused hemoglobin in the bone marrow. Evidence for this was lacking, although in some respects it was an attractive hypothesis. It should be noted that Heilmeier believed that fecal urobilinogen excretion in pernicious anemia corresponded well with the rate of decline of the total circulating hemoglobin and on this basis he assumed that it represented simply excessive hemolysis.[109] For reasons that are not clear London and West[110] have ascribed to us, rather than Heilmeier, the assumption that all of the bile pigment excreted in pernicious anemia is from destroyed hemoglobin. As noted above, our earlier observations[26,107] indicated that this was not the case.

The studies of London and co-workers[3,110] with N_{15} glycine have gone far in clearing up these questions. In following N_{15} in the fecal stercobilin of normal subjects, after administration of N_{15} glycine, it was found that there were two peaks, one very early, representing 15-20 per cent of the total stercobilin, and the other at the expected interval of 120 days, that of the life span of normal red cells. The first peak has not been adequately explained, although the general view is that it relates to the formation of excess pigment in accordance with the "pigment pool" concept of Whipple.[105] The possibility must also be considered that it represents destruction of hemoglobin which, in the sense of Jedlicka,[108] has not entered the circulation, or that it represents hemoglobin of young red cells which do enter the circulation but which are destroyed within a short time either because they represent compensation in excess, or for other reasons. It is, of course, possible that only the exact number of red blood cells are released into the circulation which are needed to replace those that have been destroyed on any given day. However, nature is lavish and often compensates in excess. By comparison with many other natural processes which are better understood, it would scarcely be surprising if an excessive number of reticulocytes were delivered each day, some shortly destined for destruction.

London and West's observations in pernicious anemia in relapse[110] indicated that about 60 per cent of the fecal stercobilin was derived from sources other than mature circulating erythrocytes. At the same time this study indicated that there was also a random destruction of circulating red cells of moderate degree, the life span being reduced to about eighty days. In view of what has already been said, it is quite probable that the hemolytic factor in pernicious anemia is less important and that a considerable fraction of the total pyrrol pigment is fabricated and not used, quite in the sense of Whipple's "pigment pool." It remains to be determined whether, because of its peculiar characteristics in respect to the megaloblastic bone marrow and maturation arrest, pernicious anemia and perhaps other megaloblastic anemias are the only conditions in which this obtains, in other words whether the smaller proportions noted in the early period in the normal

individual and in other diseases are on a different basis, as has just been considered.

Dipyrrylmethene Problem

Finally it is well to consider the possible significance of dipyrrylmethene formation, which was alluded to in the foregoing. At the time stercobilin was first crystallized from the feces in 1932[67,88] the author noted that it was accompanied by considerable amounts of a dark brown pigment of differing behavior and solubility characteristics, designated then as "copronigrin."[68] Instead of a sharply defined absorption band with maximum at 495 mμ, it presented broad diffuse absorption of ill defined character extending from about 500 mμ on down into the blue. It did not exhibit the characteristic green fluorescence of the urobilin group with zinc acetate but instead precipitated as an insoluble zinc complex. It was also observed at that time that similar characteristics were exhibited by what Fischer had earlier designated as "Körper II," a brown pigment appearing during the amalgam reduction of bilirubin to urobilinogen.[57] Although the similarity was recognized, no further studies were made, and it remained for W. Siedel[111,112] to provide detailed information as to the character of these compounds. His studies leave no doubt that the substance from feces which the author had called "copronigrin," purely in a descriptive fashion, and Fischer's "Körper II," are closely related if not identical. His analyses of the latter agreed with the formula of a dipyrrylmethene to which he gave the name "mesobilifuscin," the prefix meso simply indicating that the vinyl groups present in the original bilirubin molecule had been reduced to ethyl groups.

Siedel also isolated "bilifuscin," the dipyrrylmethene with the structure which one would anticipate if bilirubin itself were split in half. It is of interest that Städeler, who first crystallized bilirubin in 1864, observed an accompanying brown impurity to which he gave the name "bilifuscin."[113] This was repeatedly observed in the ensuing years, but Siedel was the first to identify it as a dipyrrylmethene and the immediate precursor of the mesobilifuscin which has just been discussed. Siedel isolated bilifuscin from gallstones and showed that it was readily reduced to the meso-compound. There is no reason to doubt that the bacterial flora of the colon can also effect this change, just as they reduce bilirubin to mesobilirubinogen and farther. Later, Siedel and co-workers[112] observed that the majority of the mesobilifuscin in the feces is excreted as a colorless chromogen which was named "mesobilileukan" or "promesobilifuscin." Siedel believed that there were two isomers both of the chromogen and the pigment, and that the pigment molecule polymerizes in varying degree, the size of the polymer relating to the solubility in chloroform and other organic solvents.

The fact that the bilifuscins and mesobilifuscins have not been crystallized and therefore perhaps not adequately purified induced Dobeneck[114] to question whether Siedel's conclusions, as just referred to, are entirely valid. Dobeneck doubted the conclusion that the mesobilifuscin, or "Körper II" as obtained *in vitro,* was identical with the pigment from the feces. This question cannot be answered until further purification has been achieved. Nevertheless from a practical standpoint there is little doubt that one or more mesobilifuscins accompany the urobilinogen and urobilin group in the feces. Indeed the fuscins are probably of greater importance for the brown color of the feces than are the urobilins. In the freshly passed stool the colorless urobilinogens greatly predominate over the orange-yellow urobilins and it is obvious that neither could well be responsible for the brown color. The fuscin color is extremely intense and dark and although much of the fuscin is excreted as a chromogen, a small amount of the pigment would suffice to produce the brown color.

There are a number of important questions about the dipyrrylmethenes in respect to hemoglobin catabolism. As we have already seen, the substance which Bingold designated as "pentdyopent" is undoubtedly a dipyrrylmethene derived from hemoglobin and occurring in small amounts in some urine samples, mainly as a result of splitting of bilirubin or one of its immediate tetrapyrryl derivatives. It is clear, however, that pentdyopent differs from bilifuscin or mesobilifuscin (Mbf) or their respective chromogens. The latter do not exhibit the characteristic absorption spectrum of pentdyopent nor the corresponding pink color. Pentdyopent has not been observed in the feces. Bilifuscin has been identified in bilirubin containing urines,[112] but there is much reason to believe that in this location, at least, it represents a chemical artefact from the splitting of bilirubin either in the urinary tract or after

the urine has been voided. It is not yet clear to what extent the mesobilifuscin of the feces owes its formation to a splitting of bilirubin or one of its immediate tetrapyrrl derivatives in the intestinal tract, or whether some fraction of the hemoglobin protoporphyrin or the bilirubin derived from it is split to bilifuscin prior to its excretion in the bile. Recent studies* with Dr. Sigrid Gilbertsen and Miss Hawkinson have shown that feeding of N_{15} labeled mesobilirubinogen in human subjects results in a variable degree of labeling of the fecal Mbf. On the other hand, a study with Lowry* has yielded conflicting data. No labeling of the fecal Mbf was found at the time of the second peak for stercobilin (ca. 120 days), following administration of N_{15} glycine. Marked labeling was observed, however, at the time of the first N_{15} peak of the stercobilin, a few days after the glycine was given. This is receiving further study at the present time.

If the fecal Mbf were shown to be simply a type of chemical artefact derived from the splitting of bilirubin or members of the urobilinogen group in the intestinal tract, this might well account for the discrepancy which has already been noted between the amount of bilirubin in the bile and urobilinogen in the feces per day. In any event, it is clear that the fecal mesobilifuscin is not quantitated with the urobilinogen and in the past has been omitted completely in considerations of bile pigment excretion based on fecal urobilinogen determination. Studies of this problem have been in progress in this laboratory for some time. With Dr. Sigrid Gilbertsen and Miss Hawkinson an attempt is being made to determine the urobilinogen-mesobilifuscin (U-Mbf) ratio in health and in certain disease states.* Admittedly the method used for mesobilifuscin is not precise, and because of the possibility of varying polymerization, it is impossible to be certain that the values which are obtained relate consistently to a homogeneous compound having the same optical density. Our data thus far indicate, however, that the U-Mbf ratio under normal circumstances is relatively constant and that there are significant variations under the circumstances of certain disease states. In the main the ratio in the normal feces is in the neighborhood of 10:1. In certain hemolytic states a ratio as low as 1:1 has been observed. It is of interest, though not yet explained, that in two cases of cholecystocolic fistula where the bile gained access to the colon directly, the U-Mbf ratio was about 50:1.

From the standpoint of detecting an increased rate of hemoglobin catabolism in human disease the Mbf determination has not thus far provided essentially different conclusions than those which would have been reached on the basis of fecal urobilinogen determination alone. In some instances it has thrown additional light on the magnitude of the total pigment excretion. One of the principal objectives at the outset of the study was to determine whether the value would at times provide evidence of increased hemolytic activity in certain disease states in which it has been reported that the Cr_{51} life span of the red cell was shortened despite a lack of increase of the fecal urobilinogen. Such a difference has been reported by others, especially in cases of leukemia,[115] liver disease[116] and certain other conditions. Dr. Paul Hagen, in this department, has been especially interested in comparing fecal urobilinogen with Cr_{51} life span measurements.* While it is true that these were selected cases of hemolytic disease he has not thus far observed an instance in which a decreased life span by the chromium method was not associated with an increase of the fecal urobilinogen, at least when the latter was taken into account with the total circulating hemoglobin. Capps[116] has observed several cases of hepatic cirrhosis in which the Cr_{51} life span was apparently somewhat shortened, without increased urobilinogen excretion. Dr. Gilbertsen has made similar observations in a few instances. Jandl,[117] however, reported excellent, general agreement between the two measurements in patients with cirrhosis.

Summary

It would be difficult to summarize adequately and with sufficient brevity the material that I have attempted to cover in this lecture. Figure 6 is presented in the hope that it may partially accomplish this purpose. As noted, the plan in Figure 6 does not attempt to decide whether the normal catabolic pathway from hemoglobin to bile pigment is via a green hemoglobin or hematin, or perhaps both. The same may be said of the transition of bilirubin to urobilinogen, in respect to

*To be published.

*To be published.

d-urobilinogen and dihydromesobilirubin. The concept of the urobilinogens and urobilins as a group of similar but clearly distinct substances is re-emphasized. From the clinical point of view it bilirubin and dihydromesobilirubin in the feces. Much more information is also needed as to the significance of the early peak of N_{15} in the fecal stercobilin, following administration of N_{15} glycine.

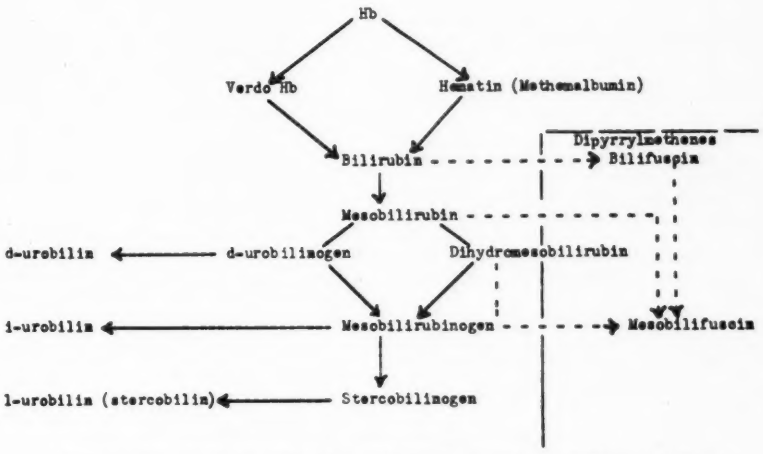

Fig. 6. Survey of the (bile pigment) products of hemoglobin catabolism.

is believed that the distinction of members of the group is not of significance in characterizing different disease states as, for example, hemolytic vs. hepatic diseases. All of the older and more recent studies in this laboratory have clearly supported the view that the urobilinogens are representatives of a stepwise reduction of bilirubin by the intestinal bacterial flora. While these studies do not exclude an hepatic formation of mesobilirubinogen, they have not supported such a concept and have indicated that it is relatively unimportant if, indeed, it occurs at all.

Recent studies of the serum bilirubin and the van den Bergh reaction, notably the observations of Cole and Lathe, fully confirm the concept of a fundamental difference between the direct and the delayed or indirect reacting types, the exact basis of which is still unknown. From the standpoint of diagnosis the value of the fractional (one minute and total) serum bilirubin determination, as employed in this laboratory for a number of years, is fully supported.

It is evident that it has not thus far been possible to cast an exact balance between the catabolic excretory products of heme and the daily wastage of hemoglobin. Further studies are needed relative to the importance of the dipyrrylmethene group and the amounts of bilirubin, mesobilirubin and dihydromesobilirubin in the feces. These are but some of the more important aspects of problems that have been considered.

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BRONCHOGENIC CARCINOMA WITH UNUSUAL PRESENTING SYMPTOMS

(Continued from Page 464)

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Current Concepts

PERIPHERAL ARTERIAL EMBOLISM

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Recent improvements in techniques of blood vessel surgery have stimulated interest in the evaluation of the treatment of arterial emboli. Although successful embolectomy dates back to 1911 when Labey[1] reported the first surgical success, the problem of treatment of arterial embolism has not yet been completely solved. Until 1934 the emphasis of treatment was upon surgery, but with the influence of Denk,[2] who introduced papaverine, of J. C. White,[3] also Leriche and Fontaine[4] who introduced lumbar sympathetic blocks and in 1936 of Murray[5] who introduced anticoagulants, the trend changed to conservatism. As one would expect, the pendulum has now swung back to a preference for surgical treatment. This has been stimulated by the ever increasing surgical interest in vascular diseases.

Survey of the literature reveals that most papers consist of reports of relatively few cases. The best overall picture of the situation is obtained from the paper by Haimovici[6] in 1950, in which he reported 330 cases of peripheral arterial embolism in 228 patients; and from the paper of Warren, Linton and Scannell[7] in 1954, reporting 337 emboli in 200 patients.

Etiology

The etiology of peripheral arterial embolism has been well established as being a complication primarily of rheumatic heart disease or myocardial infarction. Infrequently the etiology may be subacute bacterial endocarditis, pulmonary vein thrombosis, or undetermined. The site of the offending thrombus lies in the left auricular appendage in cases of auricular fibrillation. The left ventricle is the source of the embolus in cases where a mural thrombus has formed upon infarcted myocardium. The aorta is the source of the embolus in instances in which a plaque or a thrombus on a plaque has become dislodged and has migrated distally in the arterial tree. Warren reports the source as undertimed in 12.5 per cent of cases, while Haimovici states that the undetermined group make up 3.1 per cent of cases.

Site of Lodgment of Emboli

The lower extremity vessels are the sites of lodgement of emboli in more than 75 per cent of cases. Warren found the figure to be 76.2 per cent, while Haimovici states that 84 per cent of peripheral emboli occur in the lower extremity and 16 per cent in the upper extremity. The frequency with which various arteries are involved are as follows: femoral, popliteal, common iliac, brachial, aortic bifurcation, axillary, external illiac, posterior tibial, anterior tibial, radial, ulnar.

In general, this picture seems to hold true throughout various reports. Multiple emboli may occur simultaneously as "showers" or at separate times in separate locations or the same location.

Pathologic Physiology

Sudden obstruction of the arterial tree by an embolus causes several physiologic events to take place. The obstruction itself, depending upon its location in the artery, may cause the death of the limb; however, collateral circulation may be such as to maintain the viability of the limb. Lodgment at arterial bifurcations is prone to lead to gangrene for in this location the collateral channels are more liable to occlusion than when the embolus lodges in the straight portion of a vessel.

Spasm of the adjacent arterial tree may be severe and may of itself be responsible for the death of the limb. Leriche and Fontaine[4] demonstrated that the spasm originates as a nervous reflex in the damaged arterial wall, spreading via the sympathetics to produce spasm not only of the

main channels, but of the collaterals as well. Sympathetic block or sympathectomy effectively relieves this spasm.

Distal thrombosis in the arterial tree is the most difficult problem to handle in these cases. Soon after the stream of blood has been slowed by the lodgment of the thrombus, there begins development of a propagating distal thrombus which continues to the next collateral channel. If the blood flow is sufficiently impaired, the propagation continues into the distal arterial tree until it may occlude the entire arterial bed. Proximal extension of the clot may occur for the same reasons. The failure to clear the arterial tree of thrombus is the basis for failure of operative embolectomy in a high percentage of cases. Changes begin to take place in the intima of the artery at the site of the embolus soon after the lodgment of the clot. Within a relatively short time, these changes are irreversible. Studies in the dog show that the changes in the adventitia are more pronounced than in the media and intima.[8] This inflammation of the adventitia may be the stimulus which causes the vasospasm in the adjacent vessels. If changes progress to any great degree in the intima, all attempts to re-establish blood flow are doomed to failure.

Symptoms

The honored quintet of pain, pallor, paresthesias, pulselessness and paralysis cannot always be found. Most of the patients complain of sudden pain followed by numbness and coldness; however, about a fifth of the patients complain of numbness and coldness without initial pain. In a few cases, the embolism is apparently silent.

The initial pain is frequently located in the region of lodgment of the embolus. Later pain develops in the distal part of the extremity along with numbness, tingling and coldness.

Absence of pulsations is the most important sign and the site at which the pulsations disappear usually represents the location of the embolus. In some instances, vasospasm is so severe that the thrombus appears to lie proximal to its true site. Oscillometry reveals absence of oscillations distal to the embolus and increased oscillations proximal to the embolus.

Diagnosis

Diagnosis of arterial occlusion is usually relatively simple; however, the differential diagnosis between thrombosis and embolism is not always easy. The possibility of embolism is always present in the person with heart disease and any complaint referable to the limbs in cardiac patients should be attended to promptly to determine the possible presence of embolism. If one cannot differentiate between thrombosis and embolism, "it is better to err on the side of embolism, since therapy cannot wait upon academic proof."[9] Diagnosis of the site of lodgment of embolus is important in the therapy and depends upon the site of initial pain, the site of disappearance of pulses, the oscillometric readings, and knowledge of the usual sites of lodgment of the embolus. In some instances, arteriograms may be a benefit, but in general this study is not necessary.

Prognosis

The fate of the limb depends upon the site of occlusion, the degree of pathophysiologic change associated with the lodgment of the embolus, the condition of the patient, and the delay in onset of treatment. The incidence of gangrene in the lower extremity varies from 12 to 34 per cent and in the upper extremity from 3 to 7 per cent. It is often stated that embolism of the upper extremity never causes loss of limb, but statistics belie this statement. Gangrene occurs in decreasing frequency in the following list of sites of embolism:

Common Iliac	62.5%
Aortic Bifurcation	55%
Femoral	34%
Popliteal	6.6%

The high percentage of limb survival in the face of popliteal embolism prompts the adoption of a policy of non-surgical treatment rather than surgical exploration in most cases of popliteal embolism.

The overall mortality varies considerably in written reports. Warren[7] reports a mortality of 27.8 per cent in 102 cases, with 165 emboli seen from 1947 through 1953. This figure represents a reasonable average for the larger series reported.

Treatment

The treatment of peripheral arterial embolism resolves itself into two primary categories, the non-surgical and the surgical methods of treatment.

The non-surgical treatment consists of the use of vasodilator drugs, sympathetic nerve interrup-

tion, anticoagulants, and physiotherapy. Vasodilator drugs, such as priscolene or hydergine, benefit the local area by causing general vasodilatation. These should be used to the point of tolerance of the patient, but fall of blood pressure should be avoided for a decrease of pressure reduces the chances for survival of the already comprised limb. Priscolene or hydergine are used in 25 mg. or 50 mg. doses as often as every four hours. Because of variations of responses to the drug one should start with the smaller dose and increase the intake of the drug as it is tolerated by the patient. Papaverine may be used in doses of 0.03 mg. (gr. ½) intravenously or intramuscularly every two to four hours.

Sympathetic nerve interruption by blocking with novocaine is the most effective method of relieving vasospasm and promoting maximum circulation in the involved limb. Repeated sympathetic blocks are often necessary, but should not be undertaken while the patient is receiving anticoagulants. If repeated blocks are anticipated, it is wise to place a small polythene catheter adjacent to the sympathetic trunk for repeated instillation of novocaine. Surgical sympathectomy is an important adjunct treatment to be employed when one does not plan to explore the occluded artery. Sympathectomy may be done in the early stages to increase the chances for limb survival, or it may be done later to relieve the ischemic symptoms which may follow the arterial occlusion.

Anticoagulant medication is probably the most important single factor in the treatment of peripheral embolism for it acts to prevent the propagation of the clot which occludes the arterial tree beyond the site of the lodgment of the embolus. Heparin given intravenously as a continuous drip made up by adding 200 mg. to 1000 cc. of 5 per cent glucose in water is the most effective and most reliable method of interfering with the clotting mechanism of the blood. Administration of the heparin solution is started at 15 to 20 drops a minute and is controlled by checking the clotting time by the Lee-White method. The clotting time should be maintained at twice normal. When the decision is made not to undertake exploratory operation, the patient can be switched to tromexan or dicoumarol for continuation of anticoagulant therapy for a period of two or three weeks. The prothrombin time should be kept at 20 to 25 per cent of normal. Excellent results have been reported in the literature with the use of anticoagulants. Haimovici[6] reports recovery of 10 out of 12 cases, attributing the success to anticoagulants.

Physiotherapy plays little part in the recovery from acute embolism. The avoidance of heat and elevation is of paramount importance! The metabolism of the tissues is increased by heat, but the tissues are not adequately supplied with blood to maintain the increased metabolism and necrosis of tissue is enhanced. Refrigeration is unwise for it may lead to irreversible changes in the tissues. The pavex boot and the use of intermittent venous occlusion have largely been discarded as not adding to the treatment of embolized limb.

The surgical treatment consists of exploration of the artery at the point of occlusion at the earliest possible moment. The chances of limb survival are infinitely greater if operation is carried out within twelve hours of the time of embolization than if the operation is delayed longer than twelve hours. Warren, Linton, and Scannell report that embolectomy within twelve hours led to recovery in 63.1 per cent of cases, while embolectomy done between twelve hours and forty-eight hours led to recovery in only 22.2 per cent of cases.

As has just been indicated, early embolectomy is of paramount importance. Direct exposure of the artery at the site of embolus gives much greater chance for success than proximal exposure with attempt to extract clot from a distance. Gentleness and avoidance of trauma to the vessel, particularly to the intima, increases the likelihood of success. Careful closure of the arteriotomy decreases the chances of thrombosis of the vessel in the postoperative period. Linton advises occlusion of the distal artery to prevent dislodgement of the embolus. On the contrary, proximal control of the vessel to release the pressure on the embolus is of greatest importance. Proximal and distal control are mandatory after the release of the obstruction in any open vessel, but free bleeding should be obtained from both ends of the vessel at the time of arteriotomy. Routine flushing of the distal arterial tree with heparin in saline solution (100 mg. of heparin in 500 cc. saline) is carried out on this service after embolectomy, unless the backflow is very brisk from the distal vessels.

The use of anticoagulants in the postoperative period is limited to those patients in whom there is some decrease of arterial blood flow indicating

that the distal arterial tree has not been completely cleared of thrombus in spite of careful suction and milking of the leg.

For the sake of illustration we have chosen a case of embolism of the brachial artery to illustrate several points.

Case Reports

Case 1.—J. V., a white man, aged fifty-two years, had been known to have had auricular fibrillation for twelve years. In 1940, he had undergone subtotal thyroidectomy for exophthalmic goiter. In 1946, he was hospitalized for a short period of time because of congestive heart failure.

On October 11, 1953, at 8:00 P.M., while seated in his own home, he noticed sudden onset of severe pain in the upper right forearm. The pain spread to the entire forearm and to the hand, then the hand became numb and cold. At 10:00 P.M., examination revealed absence of radial and ulnar pulses. The brachial pulse was palpable to just above the ante cubital fossa. There was hyperesthesia of the hand and lower forearm, paleness and coldness of the entire forearm and hand. The veins of the forearm were collapsed.

Embolectomy was carried out at 11:40 P.M. A large firm embolus was removed from the brachial artery, just above the bifurcation. After removal of the clot, there was free flow from the ulnar and radial arteries and from the brachial artery. The arteriotomy wound was closed with continuous suture of 5-0 arterial silk, after injecting 10 mgm. of heparin into the distal arterial tree. Postoperatively, the radial and ulnar pulses were of good quality. Recovery was uneventful.

The patient was well until January 31, 1955, when he awoke in the morning with numbness and tingling of the lower half of the right forearm. The patient felt for the radial pulse and none was palpable. He came to the hospital at once, where examination revealed no radial or ulnar pulse. Sensation, motion and color of the hand were normal. The middle and ring fingers remained cooler than the rest of the hand. He was given 25 mg. of priscolene intramuscularly and 75 mg. of heparin intravenously at 8:00 A.M. By 9:00 o'clock the radial pulse had returned, and the hand "felt normal." The clinical impression was embolism of the volar arch of the ulnar artery with associated vasospasm. A good recovery from this incident resulted, except excessive use of the hand brings out early fatigability of the middle and ring fingers.

In an attempt to avert further embolic episodes, the patient was admitted to the hospital April 14, 1955, for amputation of the left auricular appendage. At operation, the heart was found to be markedly enlarged, throughout all chambers, especially the ventricles. There was no mitral stenosis. Amputation of the auricular appendage was effected and the patient's postoperative course was uneventful. To this date, he has had no further embolic episodes.

Comment.—This patient serves to illustrate both the surgical and non-surgical treatment of peripheral embolism. He further illustrates the fact that removal of the embolus at an early time affords a better opportunity for the avoidance of post-embolic ischemic symptoms than the non-operative treatment. Even though the percentage of limb survival is high in patients with brachial embolism, it is important to attempt to restore circulation to as near normal as possible, thus avoiding the post-embolic ischemic symptoms which may occur.

Case 2.—Mr. C. N., a white man, aged sixty-four years, with known auricular fibrillation, had onset of pain, numbness, coldness and pallor of the right lower extremity at noon on September 10, 1955. When first seen at 8:00 P.M., the leg was cyanotic to the mid thigh, was cold, hypesthetic and there was no palpable femoral pulse. Auricular fibrillation was present. Exploration of the right common iliac artery was carried out, and a large embolus was removed. Distal clot 4 cm. in length was removed, but free flow of blood was not obtained. Arteriotomy of the common femoral artery was done, and a thrombus 37 cm. long was removed. The tip of this thrombus was forked and pointed, indicating that it had extended into the anterior and posterior tibial arteries. The pointed ends of the clot probably indicated that the thrombus had extended distally to this point only. Poor femoral pulsation resumed after closure of the arteriotomy, and the leg remained cold and cyanotic. Postoperatively, the patient was given heparin for half an hour, then this was interrupted by a blood transfusion and heparin was not again started until four hours later. Six hours after the operation, the leg was of normal color and temperative, but the distal half of the foot was cold and somewhat cyanotic. Twelve hours after operation, the entire leg and foot appeared normal. No pulses were palpable in the foot, but the femoral pulse was palpable. The patient was discharged from the hospital a week later with a warm, viable limb.

Comment.—This patient illustrates the changes which occur when surgery is delayed as long as twelve hours after the onset of symptoms. His operation was done almost twelve hours after the onset of the occlusion. Had he been operated upon earlier, one would have been faced with less of a problem from distal thrombus. Another lesson is to be learned from this patient. The lesson is that one must persevere in attempts to clear the arterial tree regardless of how impossible the situation may seem to be.

(Continued on Page 486)

Continuation Studies

DYSPHAGIA

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Dysphagia is a symptom manifested by a great many organic diseases and functional disorders. Dysphagia is a general term used to denote some disturbance of the swallowing mechanism.

For purposes of maintaining some continuity of thought regarding the causes of dysphagia, we will first consider the upper swallowing mechanism and then the lower swallowing mechanism.

In consideration of the upper swallowing mechanism, brief reference should be made to the three stages of swallowing, of which the second stage, in which the bolus passes through the pharynx into the upper end of the esophagus, is most complicated and involved in the disturbances of the upper swallowing mechanism.

The muscles which take part in the propulsion of food through the pharynx are the muscles of the soft palate, the constrictors and elevators of the pharynx, and the muscles which close the upper aperture of the larynx. The powerful action of this system of muscles propelling food through the pharynx is referred to by Negus[1] as the force-pump action. To complete the second stage of swallowing the powerful cricopharyngeus muscle, which, during inspiration, maintains a constant spasm to prevent air from entering the esophagus, now must relax in just the right time to admit the bolus of food into the upper esophagus.

In speaking of functional diseases of the upper swallowing mechanism we generally refer to three main groups of disturbances: those due to changes in the action of the musculature, those due to interference in the innervation, and those of purely psychogenic origin. The first group comprises those diseases which cause changes in the action of the musculature without obvious local lesions or gross changes in its structure and includes: myasthenia gravis, scleroderma, dermatomyositis, the myasthenia sometimes associated with tuberculosis or thyrotoxicosis, and the myasthenia associated with advanced senility.

The second group includes disturbances of both motor and sensory innervation. The motor nerve supply to the pharyngeal constrictors and the laryngeal muscles is the vagus. The tensors of the palate are supplied by the fifth nerve and the elevators by the vagus through the pharyngeal plexus. The sensory supply to the pharynx is from the glossopharyngeal and vagus nerves.

Motor disturbances usually produce weakness or paralysis but in some instances may cause spasm. The most common causes of paresis are:

1. Peripheral neuritis such as post-diphtheretic neuritis or that following other acute infections.
2. Jugular foramen syndrome due to lesions in region of the jugular foramen.
3. Lesions affecting nerves as they leave the bulb to enter the jugular foramen such as meningo-encephalitis, tumors, fractures.
4. Intra cranial lesions such as: hemorrhage, emboli, or tumors affecting the bulb, tabes dorsalis, poliomyelitis, disseminated sclerosis, syringomyelia and amyotrophic lateral sclerosis.
5. Osteoarthritis of the cervical spine or cervical spondylosis, a very important cause of the globus syndrome and one which is too often overlooked. Morrison[2] has emphasized the need for careful analysis of the "globus patient," including x-ray examination of the cervical spine, before dubbing him a neurotic. He pointed out that the gradual degeneration of the intervertebral disc elements eliminates the normal cushioning effect, resulting in opportunities for compression of the cervical nerves. The intercommunication and referral of afferent and efferent impulses between the cervical nerves and the V, IX, X, and XI cranial nerves is well established and

affords an understanding as to the cause of dysphagia in cervical spondylosis.

A tonic spasm of the pharyngeal musculature may occur in tetanus, certain brain injuries, botulism, and in acute local infections.

The occurrence of unexplained pain in the region of the cricoid cartilage which is relieved by swallowing is presumed to be due to spasm of the cricopharyngeus.

Psychogenic disturbances of the swallowing mechanisms, which constitute the third group of swallowing difficulties, are very common and apparently on the increase as the tension of modern living mounts. The usual complaints referable to the pharynx are pain, burning, dryness, choking, and sensation of a lump in the throat. Dysphagia of pyschogenic origin varies from mild forms of globus hystericus to more severe forms actually interfering with the passage of food and associated with true neurosis, hysteria, or even psychosis.

Foreign bodies lodged in the cricopharyngeal pinch cock will of course cause dysphagia until removed. Dysphagia may result from any local injury to the pharyngeal tissues such as might occur from laceration, chemical burns, fire, smoke, gases, and other similar agents. Likewise postoperative wounds in the region may cause temporary dysphagia. Occasionally postcricoid irritation is caused by a nasal feeding tube. Tumors of the palate, pharynx, and larynx will often cause dysphagia. Postcricoid carcinoma may be associated with dysphagia early or late in its course.

Pulsion diverticulum of the hypopharynx, which we see frequently, results in dysphagia or in some instances may be precipitated by dysphagia. This herniation of the sac occurs through the weakened triangular area posteriorly where there is a deficiency of muscle fibers due to the obliquity of the inferior constrictor muscles of the pharynx. The bulge occurs when there is a failure on the part of the cricopharyngeal constrictor to relax synchronously with the powerful force-pump action of the pharyngeal constrictors.

Dysphagia may develop as a result of a few conditions existing between the upper mechanism of swallowing and the lower or cardiac mechanism. These conditions include foreign bodies; esophageal carcinoma; certain benign tumors of the esophagus such as neurofibroma and leiomyoma; extrinsic pressure due to mediastinal tumors, bronchogenic carcinoma, heart disease, abberant blood vessels, and aortic aneurysm; and benign strictures.

In approaching the lower sphincter we embrace a whole new group of disturbances causing dysphagia. Here again we see both functional and organic diseases.

Cardiospasm or achalasia occurs at the lower end of the esophagus, but its exact location, mechanism, and etiology are matters of considerable controversy. The theory, proposed by Hurst, that the spasm occurs as a result of atrophy of Auerbach's plexus seems most logical since the function of this parasympathetic nerve plexus is to cause muscle relaxation when stimulated.

Esophageal hiatal hernias of either the congenitally short esophagus or the sliding type may produce dysphagia from the regurgitant esophagitis resulting from the incompetency of the cardia. Incompetence of the cardia may also result from surgical procedures on the esophagus for cardiospasm or benign stricture. Aberrant gastric mucosa with peptic ulceration has been found in the lower esophagus. Ulceration of the esophagogastric junction following gastrectomy for peptic ulcer or carcinoma will cause dysphagia.

The differential diagnosis of dysphagia involves a careful history taking, roentgenograms of the cervical spine, esophagrams, and esophagoscopy.

Treatment may be medical, surgical, physiotherapeutic, psychiatric, or a combination of these treatments, depending on the disease. Esophageal dilatations have been very useful in the treatment of benign strictures, the globus syndrome, cardiospasm, and inoperable carcinoma. The endoscopic application of the Mackler tube has been a useful palliative instrument for inoperable esophageal carcinoma.

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Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

PSYCHOSOMATIC RESEARCH

Although the clinical psychologist is engaged in individual research with psychosomatic problems, the current trend is for him to function as a member of a team which includes psychiatrists, as well as members of other specialties, such as internists, surgeons, or gynecologists. When the clinical psychologist is involved in a psychosomatic problem individually, he is usually testing well- formulated hypotheses by means of standard psychological techniques such as the Rorschach, the Thematic Apperception Test (TAT), the Minnesota Multiphasic Personality Inventory (MMPI), or rating scales of behavior. For example, in an attempt to study the specificity of peptic ulcer in relation to intense oral conflicts, use was made of two projective techniques, the Rorschach and the Blacky pictures. It was concluded that a conflict over oral-dependency needs did not significantly differentiate a group of peptic ulcer patients from patients with nongastrointestinal psychosomatic disorders. In another individual study, an attempt was made to test the hypothesis that patients with essential hypertension show less aggression during stress than do normotensive individuals. Using the TAT in a stressful manner and testing aggressivity by means of the Allport Ascendance-Submission Study and the Rosenzweig Picture-Frustration Study, two standard psychological techniques, it was found that the two groups did not differ in aggressivity. However, some behavioral differences were noted in overt aggression in that some normotensives refused to carry on with the task under stress.

Team studies of psychosomatic disorders have been common in recent years. For example, a study was made of personality patterns of children and adolescents with peptic ulcer syndrome. Although a psychiatrist and a psychologist were the principal investigators, internists and radiologists were also involved. A series of studies utilizing a team approach have come from the Memorial Center for Cancer and Allied Diseases dealing with the psychological impact of cancer and cancer surgery. Although the findings are too numerous to mention, the psychologist's contribution was that there was no consistent pattern on psychological tests in patients who have had a colostomy, dispelling the possible notion of a "colostomy personality." In still another team study on the relationship between personality factors and human cancer, it was found that the MMPI could readily detect cancer patients with rapidly advancing disease from similar cases in which the period of survival was far longer than the average expectancy. The results suggested the possibility that long-term and intense stress might exert an accelerating effect on the rate of growth of an established cancer.

Since psychosomatic medicine is such a multidiscipline problem, as demonstrated by the numerous research studies, it seems inevitable that treatment of the psychosomatic patient should also reflect the team approach rather than be attempted by any one specialty or discipline.

MURRAY S. STOPOL, Ph.D.
Clinical Psychologist
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PHYSICIANS-HOSPITALS-PUBLIC

The medical profession is fortunate in that it can control public relations with but little effort and expense, by every doctor appointing himself a committee of one on public relations and sincerely and diligently applying himself to the task. Our daily contact with our patients who have faith in us offers the best opportunity. First of all by conducting ourselves, both professionally and morally above reproach, and occasionally adding a few tactful words for the good of the cause. It is very important that the office nurses and other assistants be instructed to be pleasant and cooperative and to use tact in their contact with the patients.

We can govern our own behavior and that of our office staff but not the personnel of the hospitals. Perhaps due to the shortage of beds and the constant demands on them for entry to the

Third in a series of editorials on psychosomatic research.

hospital, they have become so arrogant that they seem to enjoy heaping insults and abuses on everyone they contact. Recently a member of my family was hospitalized in Minneapolis. She has nothing but praise for the care she received, but I have never before been subjected to such arrogance and disrespect as I received at the business office.

A short time ago I made the final payment on a hospital building fund, a year before this payment on the pledge was due. This was accompanied by a friendly letter in which I wished them success in their big venture. I did not receive a reply, not even a formal receipt.

My own experience is of no concern but, if a doctor who usually commands a little respect around the hospital receives this kind of treatment, there can be no question what kind of treatment is doled out to lay persons.

Our hospitals are so closely interwoven with the profession in the minds of our patients, both professionally and socially, that our patients think of us as a single unit.

We know that the hospitals have many difficult problems, but this does not excuse them for their anti-social attitude. If this treatment of the public by the hospitals continues, they will drag us down into socialized medicine no matter how hard we try to avert it.

F. H. MAGNEY, M.D.

TELEVISION AND THE YOUNGSTERS

Juvenile delinquency, within the short space of three decades, has been blamed on everything from phases of the moon to broken homes—including moving pictures, comic books, prosperity, and poverty. If a youngster is doing poorly in school, or misbehaving generally, the easy answer is to pick out one cause and blame everything on it. Television is handy, and no one can deny youngsters are fascinated by this magic window on the world.

But the easy answer is rarely the complete answer. Intelligent parents must take into consideration the multiple influences affecting their children. It boils down to charting individual courses on a basis of deep affection, common sense, and faith.

From Mother Goose through Kon Tiki, youngsters of all ages have thrived on adventure yarns. Some of them are terrifying (or have you forgotten "Snow White" or "Little Red Riding Hood"?). Television is aware of this interest in dramatic adventure stories, and answers the demand—with careful de-emphasis of the terrifying, the violent, and the bigoted.

This is not to say there is no conflict in television dramas. A plot requires conflict, expressed on television as a visual clash between ideas or people. To remove this plot-conflict would lead ultimately to programming without viewers. Furthermore, a gripping plot resolved with virtue triumphant—the rule on television—may very well serve to relieve repressed emotions of young viewers. If a child is secure in his home relationships, there's considerable doubt a rip-snorting Western on television will send him off on a destructive tangent.

No conscientious parent would permit his children to subsist entirely on a diet of candy—no matter how loudly the youngsters might clamor for it. Many thoughtful fathers and mothers feel similarly about television. They realize their children need plenty of sleep, exercise, and regular hours for eating. There is a definite place in the normal child's routine for reading, school work, and household chores. A little thought works out a schedule which can include these, as well as a selected television schedule.

What is TV's effect on children? A Minnesota State Medical Association committee reported in 1955 it could find no relation between delinquency and the time children spend watching television. Nor is the amount of time children spend watching TV, the committee continued, related to intelligence or to scholarship.

The television industry realizes its responsibilities to its viewers—young and old. TV programming is constantly examined and re-examined, to make it better and more appealing not only to the children, but to every member of the family.

GENE GODT.
WCCO Radio and TV

REHABILITATION IN PULMONARY TUBERCULOSIS—Its History

The first conception of the proper rehabilitation of the tuberculous came from Dr. Hermann Biggs of the United States in 1910, when he stated that

Second of a series of editorials on pulmonary tuberculosis.

"what is needed is an industrial colony, where proper occupation can be provided under proper conditions, with proper living quarters, where 'arrested' cases can earn a livelihood and maintain their health."

From this idea there originated two organizations in the same year, 1915. One was the Altro Workshops of New York, the other the Cambridgeshire Tuberculosis Colony founded by Varrier-Jones in the village of Bourn, and moved later to Papworth. Neither followed the ideal of Hermann Biggs in full detail. The Altro Workshops interpreted "arrested cases" as sputum-negative cases with minimal extent of lung damage, and while it provided sheltered employment it did not supply living quarters. Varrier-Jones provided both work and homes, and he admitted men in all stages of disease. He did not confine himself to the non-infectious, and preached the then heretical idea that the rightful place of the tuberculous bread-winner was to live with his family in a house where he would have a room to himself, which he would use continually if he remained sputum-positive, or occupy at any time he had an intercurrent illness or a frank re-exacerbation of his disease.

On both sides of the Atlantic these ventures must have caused considerable surprise; certainly both met with little help at first, and sometimes with active opposition. After all, it was only four years previously that the National Insurance Act in Great Britain had made sanatorium treatment, then hailed as the long-sought cure, available to all sufferers from pulmonary tuberculosis; and it was not until four years later, in 1919, that Dr. Noel Bardswell published the first disturbing statistics from King Edward VII Sanatorium, Midhurst, on mortality following such treatment.

But Varrier-Jones had already seen that something more was necessary than a temporary regimen of rest and graduated exercise, with good food, in hygienic surroundings. He was convinced that these temporary conditions should, for most cases in those days, be made permanent, and time proved his thesis correct as against the answers of other workers to the new problems raised by Bardswell's report. He did not condemn sanatorium treatment, because he saw in it a regime with a good middle but poor supporting ends; he realized that at one end eighty per cent of admissions were already beyond the early stages of disease, while at the other end, the sanatorium was powerless to see the implementation of the advice it gave to the patient on his discharge. He therefore built round the sanatorium as an essential section of his settlement; from it his patients were to proceed to work and to colonization, and to it they could return if further treatment proved necessary.

Any practical interest in the extension of proper services for the tuberculous has sprung from these two sources, although little more was done before the end of World War II.

R. R. Trail, M.D.
London, England

THE SYMPTOMS OF ALCOHOLISM

The range of I.Q. scores for alcoholics is the same as for non-alcoholics.

The alcoholic tends to be suspicious and sensitive, unable to tolerate pressure. He is worried, anxious, very easily depressed, and is likely to change jobs often. Another characteristic of the mature alcoholic is social isolation. He thinks that no one understands him, especially those who are close to him. He generally has very few real friends.

The alcoholic's motive for drinking is to escape the reality of life and to create a world of fantasy—unreality. He finds himself drunk regularly. He needs that morning drink to get organized.

Going on "benders" usually marks the beginning of the chronic phase. A "bender" is a period of a day or more in which a person drinks blindly, helplessly, with just one purpose—to get drunk.

Possible impairment of his character and personality traits may occur, as for instance:

Initiative—Loss of ability to start new activities.

Trustworthiness—Loss of confidence in one's sense of responsibility in work and social relationships.

Personal appearance—Resulting in poor grooming and slovenliness in dress.

Perception and memory disappear as restraint and self-control are lessened.

The field of observation is narrowed, muscular co-ordination is weakened, and clumsiness takes the place of precision, often resulting in mishaps.

Alcohol does not cause organic damage of any cells in the brain or the rest of the body; it affects only the functions of the cells as they are controlled by the central nervous system.

Fifth in a series of editorials on alcoholism.

The alcoholic is sick, for excessive use of alcohol may bring about personality deterioration and mental defects, loss of physical vigor, and nutritional deficiency.

PAUL S. RAHNEFF
Executive Secretary
Citizens' Commission on Alcoholism of Minnesota, Inc.

THE HISTORY OF GLASS CONTAINERS

Not only was the Jamestown glass plant *America's first factory,* and *America's first industry* —existing as it did a dozen years before the arrival of the Pilgrims at Plymouth in 1620—but glass was part of the first cargo ever exported from these shores.

In his "Historie of Virginia," Captain Smith wrote: "We sent home ample proof of pitch, tar, glass . . . " Fragments of glass found among the ruins indicate that bottles, vials and drinking glasses were produced at the Jamestown plant and exported to England, and it is probable that glass beads given Indians in exchange for land were produced at America's first factory.

After Captain Smith left for London in the fall of 1609, sickness, hunger and death struck the Jamestown colony in full force. Work at the glass plant first slowed down, and then stopped altogether. Twelve years later—in 1622—a Captain William Norton arrived in Jamestown with a group of Italian glassmakers, and operations were resumed. The fires of the Jamestown plant died out permanently, however, in 1624.

In 1632, two Salem, Massachusetts, colonists started a plant to make glass bottles to transport New England rum and cider to the Carolinas and the West Indies. This venture was more successful than the original Jamestown plant, continuing until 1670. And glass plants were also operating in New York and Pennsylvania during the latter half of the 17th century.

One of the most successful of America's early glass factories was founded in 1739 in Salem County in southern New Jersey, by German born Caspar Wistar, one of the great glassmakers of all time. At first, Wistar made bottles which he sold to traders who were smuggling West Indian molasses into the Colonies in violation of British regulations. Later, Wistar and his son also produced bowls, drinking glasses, preserve jars and dishes.

The Wistars are credited with making America's first flint glass, the remarkably clear and brilliant glass which was perfected in England by George Ravenscroft in 1675. And they produced, as well, magnificent colored glass. Shades included beautiful greens ranging from aquamarine to deep emerald, and ambers from dark brown to yellow and honey. Martha Washington owned a tiny Wistar scent bottle of yellow and turquoise—and probably followed the then-current fashion of carrying it tucked inside her glove.

By the time the Wistar plant closed in 1780 after more than forty successful years, another great glassmaker—William Henry Stigel, had come and gone. Called "Baron" because of his flamboyant habits, Stigel operated in Lancaster County west of Philadelphia. Stigel's ambition was to prove that beautiful glassware could be produced here as well as in Europe, and prove it he did.

England had recently passed new tax laws. British glass, along with tea and other British products, now cost the colonists dearly in import duty. These duties, Stigel concluded, were full reason for expanding and perfecting glassmaking in America. Money—even if it had to be borrowed—was no deterrent, and the result was glassware highly prized today by museums and collectors everywhere.

The flamboyant "Baron's" enthusiasms were more than his deficit financing could support, however, and, in 1774, Stigel landed in debtor's prison.

JOHN M. FOSTER
Foster-Forbes Glass Co.

PHONOCARDIOGRAPHY: A LOST OPPORTUNITY

The role and clinical applications of phonocardiography are still controversial. Unfortunately, many of those who oppose a systematic use of this method know little about it or have an incomplete knowledge of the physical principles involved.

Auscultation and phonocardiography are two inseparable approaches to the study of the same clinical phenomena. However, phonocardiography has a wider scope because it can be used either for confirmation of clinical auscultation or as a

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Third in a series of editorials on glass and glass containers.

more perfect method of study of the sounds and murmurs.

The human auditory system is highly imperfect. Its characteristics render it far better able to evaluate the high-pitched notes of a musical instrument than the lower pitched notes of the heart. Easy fatigability, sensitivity which is poor for low notes and then sharply increases on a logarithmic scale, inability to separate vibrations, difficulty in timing certain sounds, conspire to render auscultation a difficult art. Unfortunately, even the best "artists" cannot surmount certain physical and physiological limitations and frequently give contrasting opinions.

Technique.—Phonocardiography is now based on electric recording and electronic amplification, high film speed, and use of other simultaneous tracings (ECG, jugular tracing, low frequency tracing of the precordium) for timing certain waves.

A quiet room, a reliable microphone, a correctly placed chest piece, and a rubber strap to hold the microphone, are necessary. Exclusion of extraneous noises is secured (auscultatory control!) and the respiratory noises are avoided by inducing apnea.

Many of the currently presented tracings reveal improper technique, low film speed, or an obsolete method which does not allow for correct selection and amplification of the important sounds and murmurs.

Recent Developments.—Among the contributions of recent years, the following should be kept in mind:

1. Separation of various bands of vibrations by different filters (Mannheimer, 1939).

2. Description of stethoscopic phonocardiography (Rappaport and Sprague, 1941). This method allows for correct reproduction of sounds and murmurs, including vibrations at the lower limits of audibility (infra-sounds). The tracing gives an excellent reproduction of gallop sounds, snaps, and rough murmurs.

3. Description of logarithmic phonocardiography (Rappaport and Sprague, 1942). This method gives a fair correlation between sound tracings and auscultation. It also permits a fair reproduction of high-pitched sounds and murmurs.

4. Electric calibration of sounds and murmurs (Wells, Rappaport and Sprague, 1949) which supplies a rough comparison between tracings.

5. Clinical calibration of sounds and murmurs (Luisada and Gamma, 1955) permitting a more accurate comparison between tracings.

6. Selective phonocardiography (Richmond, Aravanis and Luisada, 1956). This new method has been developed for reproduction of either low-pitched murmurs, like that of mitral stenosis (first filter: emphasis on vibrations in the bands 60-110 per sec.) or high-pitched, soft murmurs, like that of aortic insufficiency (second filter: emphasis on vibrations in the bands 150-200).

Phonocardiography is not widely used because it requires special training and, except in special cases, personal study by a physician. This deprives many complex cardiac cases of one important aid in the workout of their diagnostic problems.

Phonocardiography is extremely useful for the study of patients with rheumatic valvular defects or myocardial damage, particularly for those cases where a differential diagnosis between the two groups is necessary. It has already given interesting data in congenital lesions. It correctly reveals phase, "shape," and pitch of murmurs; phase, nature, and shape of snaps, clicks, and gallop sounds. In addition, phonocardiography is the best method for timing the waves of certain tracings (ballistocardiogram, electrokymogram, intracardiac pressure tracings). An electrocardiogram is only a poor substitute for it because of variable relationships between electric and mechanical phenomena and poor definition of the end of systole.

A. A. Luisada, M.D.

SAFEKEEPING

Most large banks maintain Safekeeping Departments. Such a department can be of valuable service to you because it offers protection and safety for your stocks and bonds on a readily accessible basis, subject to your instructions. Placing your securities in safekeeping is very simple. You merely bring them to your bank's Safekeeping Department where you will be given a receipt for your securities. The department staff will prepare the necessary records, files and cross files, and will put your securities in its cash vault.

Safety and *convenience* are but two advantages in having your securities in a bank's own vault.

The Safekeeping Department staff will be glad to clip coupons from your bonds as they become due and deliver them to the bank's Collection Department. This department can collect the coupons for you and either credit your account or send you a check for the proceeds. Similarly, if a bond becomes due, the bank can collect it for you, and either send you a check or credit your account with the funds. Stock dividend checks can also be sent by the respective companies to your bank for your credit. Such servicing of your securities while they are in safekeeping at your bank will be a genuine convenience for you.

Your securities, of course, are *available* to you any time you want them. It's as easy to take them out of the bank as it is to deposit them. Simply surrender your safekeeping receipt, sign a release, and in a few minutes the securities will be transferred from the bank's vault into your hands.

Whether you are in or out of town, your securities are available to you at all times, for they are always subject to your instructions which will be immediately carried out. If you should be out of town for several months, perhaps engaging in research, studying abroad, or enjoying an extended vacation, you need have no concern for your securities if they're in safekeeping at your bank. Should you wish to dispose of some—or sell or trade them—all you have to do is to notify the bank what your wishes are, and they will follow your orders.

The nominal charge made for a bank's safekeeping service is most moderate for the satisfaction you will have in knowing your securities are safe, serviced, subject to your instructions, and always immediately available to you.

JOHN L. SANKOVITZ
Manager, Safekeeping Department
First National Bank of Saint Paul

PERIPHERAL ARTERIAL EMBOLISM

(Continued from Page 478)

Conclusions

The operative treatment of arterial emboli generally leads to better results than the non-operative treatment. Early surgical intervention is the most important single factor in the operative treatment. General heparinization is an important adjunct treatment in those cases in which it is felt that serious bleeding will not result from heparinization.

Summary

The treatment of arterial emboli is reviewed. Discussion of the various modes of therapy is undertaken with the author's recommendations as to specific details of therapy.

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HORMONES FOR SHOCK

When all other measures fail, treatment with adrenal hormones may prove life-saving in some cases of shock following acute coronary thrombosis, according to Dr. Benjamin Manchester of Washington, D. C.

The cardiologist told of six patients in whom a state of shock had persisted for several days without responding to standard treatment. Laboratory tests showed that the adrenal glands were not functioning adequately.

Dr. Manchester recalled that similar observations—of depressed adrenal function—had been made in acute stress due to other organic diseases, and that recovery had occurred when appropriate adrenal therapy was used to overcome this deficiency.—B. MANCHESTER, *The Medical Way*, 18:6 (Mar.) 1956.

Medical Economics

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Minnesota State Medical Association
George Earl, M.D., Chairman

RESULTS OF PHYSICIANS' SOCIAL SECURITY POLL ANNOUNCED

On May 8, 1956, a letter, accompanied by a post-card ballot, was sent to all members of the Minnesota State Medical Association requesting their opinions on social security coverage for members of their profession. The doctors were asked whether they favored compulsory, voluntary or no coverage under social security.

A total of 3,366 letters was sent out. By May 15, the response totaled 1,908 (nearly 58 per cent).

A report dated May 16 and submitted to the House of Delegates at the 103rd annual meeting in Rochester reported that 233 (12 per cent) of the doctors who responded favored compulsory coverage; 1,155 (61 per cent) favored voluntary coverage and 530 (27 per cent) opposed any type of social security coverage.

Responses from the metropolitan areas of Duluth, St. Paul and Minneapolis totaled 917; 779 came from small towns and 212 from Rochester. One hundred seven city doctors favored compulsory coverage compared to eighty-four in smaller towns and thirty-two in Rochester.

City doctors preferring voluntary coverage totaled 580 with 435 in smaller towns and 140 in Rochester agreeing.

Opposition to any form of social security coverage for their profession came from 230 doctors in metropolitan areas; 260 in smaller towns and forty in Rochester.

The age brackets broke down this way: A total of 343 doctors under thirty-five responded to the poll; 530, from thirty-five to forty-four; 469, from forty-five to fifty-four, 300, from fifty-five to sixty-four and 266 over sixty-five years of age.

Sixteen doctors under thirty-five favor compulsory coverage while 225 in the same age bracket chose voluntary coverage and 102 are opposed to any social security coverage.

Fifty-nine physicians between ages thirty-five and forty-four want compulsory coverage; 316 prefer voluntary coverage and 155 disapprove of any social security coverage for physicians.

Compulsory coverage is favored by sixty-six doctors from ages forty-five to fifty-four; 309 in the same age bracket favor voluntary coverage and ninety-four want none.

In the fifty-five to sixty-four age group, forty-six favor compulsory coverage; 172 prefer voluntary and eighty-two are opposed to any social security coverage for physicians.

Thirty-six doctors over sixty-five want compulsory social security coverage while 133 in the same age bracket prefer voluntary and ninety-seven want none.

Since this report was prepared, an additional 319 cards have been received indicating the same strong preference for voluntary coverage. These additional replies bring the total response to the poll to over 65 per cent, a larger percentage of replies than in the majority of other states conducting similar polls. This is a credit to the physicians of Minnesota—to the interest and co-operation demonstrated by this prompt and unusually large response.

HOUSE OF DELEGATES PASSES SERIES OF IMPORTANT RECOMMENDATIONS

The 1956 House of Delegates of the Minnesota State Medical Association passed several vital recommendations during its annual session in Rochester. Briefly, several of these measures were:

1. *A recommendation* officially requesting Blue Shield to change its corporate structure to include all participating doctors who wish to do so, to become members of the corporation. The change was recommended by the Council on petition of the St. Louis County Medical Society. Blue Shield now has only twenty-one corporate members who elect a board of eleven members.

This House action is in no way binding on Blue Shield. No action was taken on proposals that Blue Shield extend its coverage to office calls although the St. Paul Society of Internal Medicine has gone on record opposing such extensions.

2. *Approval of a resolution* calling for the establishment of a medical examiner system to supplement, not to replace, the present coroner system in Minnesota. A medical committee is to work with a Minnesota

State Bar Association committee to prepare a bill for introduction in the 1956 legislature.

3. *Adoption of a resolution* opposing the practice of medicine by corporations in Minnesota. This resolution emphasizes the fact that this applies to non-profit as well as to profit-making corporations.

SENATE COMMITTEE ACTS ON H.R. 7225

On May 25, the Senate Finance Committee forwarded the social security bill (H.R. 7225) to the Senate floor. The committee reported the bill without the major provisions in the House-passed version. Cash disability benefits were rejected; tax increases were also rejected. The retirement age for women was lowered to sixty-two for widows only.

The bill went to the Senate with no further amendment except the extension of the 1952 McFarland amendment which increases the proportion of public assistance payments paid by the government. Medical payments as a separate category under public assistance with the states getting up to $4 of U. S. money per month for adults and $2 for children have been incorporated into the social security bill. Funds would have to be matched 50-50 by the states.

MILITARY DEPENDENT MEDICAL CARE BILL PASSES BOTH HOUSES

The new military dependent medical care program sets up the financial machinery for furnishing private medical care to hundreds of thousands of wives and children of servicemen. This plan makes medical care a statutory right, defines dependents and provides uniform benefits for Army, Navy, Air Force, Marine Corps, Coast Guard and Commissioned Corps of Coast and Geodetic Survey and U. S. Public Health Service. This protection for dependents is designed to make military careers more attractive and to help reduce personnel turnover.

WHO ASSEMBLY REINSTATES RUSSIA; REDUCES HER BILL

Under terms of a compromise resolution, Russia and eight satellite countries can resume active membership in the World Health Organization by the settlement of their over-due assessments. The ninth World Health Assembly meeting in Geneva voted 51 to 0 (with five abstentions) to restore the rights and obligations of active membership provided contributions are paid in full for the years when these members participated actively in the work of WHO and five per cent of the amount assessed during inactive years. Complete payment is due within ten years.

ARE DOCTORS REALLY OVERPAID?

A recent article in "The Medical Way" reports on a comparison made between the earnings of a typical doctor and a representative bricklayer for a period of twenty years. After a two-year apprenticeship and a period of being a journeyman the bricklayer's total income for his first ten professional years is about $66,976.

A typical doctor's income for his first ten dedicated years is $5,000—none from his eight years in college and medical school and $2500 for each year of his residency. If he begins practice in a prosperous American community at the end of his residency he can expect to average earnings estimated today at $15,000 per year for the next ten years.

At the end of twenty years in *his* profession, the bricklayer has earned a total of nearly $140,000; the doctor, $155,000. However, if the bricklayer worked the average doctor's 56-hour week, he would earn an annual salary of over $12,000, a fraction less than a doctor's for the same number of hours of work.

"However," states the article, "the purpose of this comparison is not to stir up an argument over the bricklayer's pay. He is a skilled and respected artisan and is certainly entitled to whatever he earns."

MINNESOTAN RECEIVES APPOINTMENT

Dr. Robert N. Barr, secretary and executive officer of the Minnesota Board of Health, is on a nine-member advisory committee to aid in improving and extending medical care and general health services to American Indians.

WMA ACTS AS INTERNATIONAL VOICE OF ORGANIZED MEDICINE

The World Medical Association has become a strong factor in protecting and promoting the professional interests of the medical profession and the cause of world peace. Now in its ninth year, WMA is a federation of the most representative national medical associations in fifty-two

countries. The AMA is a leading member. Every individual physician in the U.S.A. is eligible for membership in the United States Committee. Annual membership dues are $10.00.

FOREIGN MEDICAL SCHOOL GRADUATES TO BE EVALUATED

At a recent meeting, the AMA Board of Trustees approved a long-sought program for evaluating foreign medical school graduates. The proposed program is designed to clear up two important facts regarding each foreign medical graduate who wishes to come to the United States as an intern or resident or in any other position that involves medical care of the American public:

1. The medical credentials of a foreign-trained physician must clearly indicate that he was graduated from a bona-fide medical school and
2. By examination, a foreign medical graduate must demonstrate that he has medical knowledge equivalent to that demanded of graduates of schools in the United States.

More than 6,000 foreign physicians are in the United States this year to complete their professional training as interns or residents. They come from eighty-four different countries and are located in all states except Idaho, Nevada, New Mexico and Wyoming.

AMA CLOSES PRINTING PLANT

June 30 marked the end of an era in American Medical Association history. That was the date the last copy of *The Journal AMA* was published in the Association's own printing plant. In its nearly seventy years of operation, the shop printed enough journals to circle the equator twice if they were laid end to end.

Beginning July 1, *The Journal* will be printed by the McCall Corporation, Dayton, Ohio, which has printed *Today's Health,* the AMA's popular health magazine, since 1950.

ADMINISTRATION SPONSORS LEGISLATION FOR MENTAL HEALTH STUDY

The administration has sponsored legislation to authorize the Public Health Service to make special project grants for the support of investigations, experiments and demonstrations in the field of mental health with particular emphasis on projects designed to improve the operation and administration of state institutions for the care or treatment of the mentally ill. The Department of HEW will request $1,500,000 this year for such grants if Congress approves this legislation.

VA TO INVESTIGATE VALUE OF "TRANQUILIZING" DRUGS

The Veterans Administration is launching a large-scale research project to investigate the value and usage of the new tranquilizing drugs for mental illness. The resources of the National Research Council will be used in planning and executing the project.

PHYSICIANS TO BE DRAFTED FOR NAVY DUTY

The selective service headquarters this month is filling a Defense Department requisition for 380 physicians for Navy duty. Data indicate that the order can be filled without tapping any doctors in Priority III past age thirty. Neither the Army nor the Air Force is expected to call for any physicians before the last quarter of 1956. Commissioning of a large crop of interns in July will help meet replacement requirements.

DOCTORS TOP EARNERS AMONG PROFESSIONS IN CANADA

In Canada where income statistics are released periodically by the Department of National Revenue, physicians top the most recent list among the professions. Their average annual income is reported to be $11,258. Close behind them were consulting engineers and architects with average incomes of $10,289.

Mortality is not the real yardstick to measure the importance or judge the control of a disease. Even if there were drugs capable of preventing the sudden deaths resulting from hypertension and arteriosclerosis, these conditions would remain a tremendous medical and social problem. Similarly, adults do not commonly die of mental disorders, arthritis, or peptic ulcers. Yet, no one would claim that these afflictions have been conquered. Neither has tuberculosis been conquered. Instead, the forces which have been at work during the past century have slowly converted it from a killing to a chronic disease.—RENE J. DUBOS, Ph.D., National Tuberculosis Association Transactions, May, 1954.

President's Letter

THE GOLDEN AGE OF MEDICINE

EDITOR'S NOTE: The following message is an excerpt from an address Dr. Wilson delivered at the annual banquet of the Minnesota State Medical Association May 22, 1956, in Rochester. Its theme was so well-chosen and well-received that we felt it deserved repeating.

You and I have had the privilege of living in a wonderful generation, a period of scientific discovery and progress such as the world has never known and, one is tempted to say, such as the world will never know again.

Medicine, as we know it, has also had its golden age in the last half century. I knew the medicine of 50 years ago because I was brought up in a doctor's family. My father grew up as a farm boy, started a sort of "hang-around" apprenticeship with a nearby country doctor and later went to medical school in Chicago for about one year. He then returned home to start practicing medicine.

On the day that I was born, April 30, 1895, his total business was $1.00, and that was charged.

Those were the days of bromidia for the nerves, calomel for the bowels, tincture or infusion of digitalis for the heart, creosote and heroin for the cough and morphine for pneumonia. Those were the days when people died of inflammation of the bowels instead of living with appendectomies. Those were the days when a woman stayed in retirement when she was pregnant. Those were the days when doctors diagnosed diphtheria by the smell. There were no x-rays; fractures were diagnosed and reduced by touch and you bought your spectacles from the grocery store.

Now let us consider in contrast what progress has been made in the past half century.

In the field of public health, for example, we have the protection of the Pure Food and Drug Act passed fifty years ago; everyone in Minnesota drinks Grade-A pasteurized milk, and I am proud to say that my home city of Winona was the first in the country, to my knowledge, to pass an ordinance requiring pasteurization of all milk sold in the city. We have strict standards for municipal water supplies;

we have public health nursing in most counties, cities and schools. And I cannot leave public health without paying a well-deserved tribute to our late beloved Dr. Chesley, a man with an outstanding national reputation in his field.

To go on to other fields: Can you imagine trying to practice medicine today without the diagnostic x-ray, the chest studies, the gall bladder, kidney, stomach and colon studies? Then, too, we have blood typing and cross-matching for safe transfusions; we have the Wangensteen suction for postoperative ileus and small bowel obstruction; we have the modern treatment of pulmonary tuberculosis with the new drugs and improved surgery.

We have modern laboratory procedures, blood chemistries, electrolytes and fluid balance. My father never heard of such things.

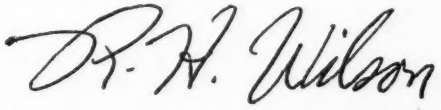
During the past half century radical changes have also taken place in medical education. The quack schools and diploma mills have gone out of existence. Continuation courses and post-graduate opportunities have multiplied until no man lacks unlimited opportunities to keep in step.

It has been a golden age of medicine, and progress is continuing at such a terrific pace that I am sure it is taxing the mental capacities of all of us to keep up with it.

I am reminded of a lovely lady of ninety-three years who sustained a fracture of the hip some twenty years ago. While convalescing in the hospital, she did a lot of reading. She would start daily with her Bible, then the morning paper and a good book and so on. She developed some eye strain, and I cautioned her about reading too much. She eased off a bit but again in a week or two she had developed quite a conjunctivitis, and I became firmer with her and told her she must ease up on her reading or I would be forced to take all the reading material from her room.

At ninety-three she looked up at me with a little twinkle in her inflamed eyes and said, 'But, Doctor, I don't want to grow up ignorant.'

Yes, with this golden age for our profession, we'll have to continue to read and read until our eyes fatigue, if we too are not to grow up ignorant.



President, Minnesota State Medical Association

History of Medicine in Minnesota

THE MEDICAL HISTORY OF RICE COUNTY
(1855-1901)

ADOLPH M. HANSON, M.D.
Faribault, Minnesota

(Continued from April issue)

Poehler, Franklin T.—He graduated from the College of Medicine and Surgery of the University of Minnesota in 1896 and was licensed in the same year. He was practicing in Morristown in 1898. He occupied an office in Minneapolis (410 Syndicate Arcade) in 1900 and continued to practice in Minneapolis for many years. He was a member of the Hennepin County Medical Society and the Minnesota State Medical Association.

Pringle, Alexander F.—He graduated from the Medical Department of McGill University at Montreal in 1880. There was a Dr. Pringle, well-known as an eye, ear, nose and throat specialist, long established at Northfield, when the writer came to Faribault in the summer of 1912. He is listed as being in Northfield as early as in 1886. He was licensed on March 10, 1885.

Ricker, George E. (Edwin).—A graduate homeopath, Hahnemann Medical College, Philadelphia, in 1878. He was practicing in Northfield in 1886. He had been licensed November 26, 1883. From 1890 through 1905 he is listed in Minneapolis and in 1909 in Washington, D. C.

Roberts, Thomas W.—Practiced in Faribault between 1886 and 1894 as the successor of Dr. Courtland B. Rockwell with an office at the corner of Main Street and 8th Avenue. He had graduated from the Hahnemann Medical College and Hospital, Chicago, in 1884 and was licensed in Minnesota April 15, 1887. It seems he had practiced for many years in Winona County where he appeared as early as 1866, before he graduated from the medical school.† There is some confusion as to the places where he practiced. Directories list him at Winona from 1890 through 1898. When he left Faribault in 1894 he supposedly moved to St. Paul, though medical directories list him there from 1903 to 1909.

Robilliard, Walter Henry.—He was born at Racine, Wisconsin, on June 20, 1861. He was a graduate of the public schools of Racine and of Lawrence College, Appleton, Wisconsin. In 1884, he graduated from Hahnemann Medical College, Chicago. He started practice at Port Huron, Michigan, in 1885. He removed to Belleville, Michigan, and from there to Liscomb, Iowa. At Liscomb he was married to Miss Eudora Joette Hauser. Three children were born to this union: Charles Morton, Harold Shaw, and Nellie. His eldest son, Dr. Charles Morton Robilliard ("Doctor Charlie"), is still in active practice in Faribault at the age of sixty-six (December, 1954). Dr. Walter Henry Robilliard was physician to the Minnesota State School for the Deaf for thirty-five years where he was respected and loved.

†See "Medical History in Winona County," MINNESOTA MEDICINE, p. 581, August, 1940.

He was Faribault's Health Officer for some years. He was prominent in civic affairs, those of the Rice County Medical Society and the St. Lucas Hospital Staff, and in Masonic circles.

He was a fiery and very positive individual who loved an argument at our Rice County Medical Society's meetings, following adjournments, with Martin L. Mayland, W. H. Rumpf, and Patrick A. Smith, F. S. Warren, and F. R. Huxley. The always unruffled Huxley was invariably the one to pour oil on the troubled waters, which were never more than tempests in a teapot, actually. The writer, for one, would not have missed one of those stormy sessions, which were a mutual friendly agreement to disagree about something, or other, to liven us all up with the recountals of mistakes in diagnosis and the loss of a patient to one of his competitors in the old days as a consequence, et cetera. Dr. Walter Henry Robilliard died in the crisis of a lobar pneumonia on November 1, 1931.

Rockwell, Courtland B.—Graduate of the Homeopathic Medical College of Chicago in 1883. He was licensed to practice in Minnesota on December 31, 1883 and practiced at Faribault for three years (he is listed there in 1890). From 1895 through 1909 he is listed in Chicago. His practice in Faribault was taken over by Dr. Thomas W. Roberts.

Rogers, Arthur C.—Born at Decorah, Iowa, on July 17, 1856; died at Faribault, Minnesota, on January 2, 1917. He received his early education in the public schools of Ohio, the Raisin Valley Seminary near Adrian, Michigan, and Barlham College, Richmond, Indiana, from which he graduated with the degree of B.S. in 1877. (This same College conferred the honorary degree of LL.D upon him in June of 1905). He graduated from the Medical School of the University of Iowa in 1883. He was clerk and physician of the Government Training School for Indians at Chemawa, Oregon, in the latter part of 1883 and the early part of 1884. While there he received his appointment as Superintendent of the State School for Feeble-Minded at Faribault, Minnesota. He entered upon these duties on August 20, 1885. He was president of the Rice County Medical Society in the years 1909 and 1910 (serving two consecutive terms).

On July 17, 1882, he was married to Miss Phoebe Coffin, daughter of Mr. and Mrs. Walter Coffin of Fort Dodge, Kansas.

His parents were Ansel and Cynthia (Benedict) Rogers, who went West and settled in Iowa in 1852. His father was a miller and a Society-of-Friends clergyman. The mother of Dr. Rogers died near Ashley, Ohio, in 1863; his father in Richmond, Indiana, in 1873.

His obituary, which appeared in the *Journal of Psycho-Asthenics,* Vol. 21, Nos. 3 and 4 (March and June, 1917), reads:

"ARTHUR C. ROGERS, M.D., LL.D., superintendent of the Minnesota School for the Feeble-Minded and Colony for Epileptics almost since its founding, and editor of the *Journal of Psycho-Asthenics* from its beginning in 1896, died January 2, 1917. . . . To the service of its editor, to whose efforts alone its existence was mostly due for many years, is due in a large measure whatever progress has been made in America, as well as the much-widened popular and scientific interest in the feeble-minded today."

This short and terse obituary says it all in a few words, but it will fail to do him full justice with the hurried or casual reader. Dr. Arthur C. Rogers was a leader and a real authority in his chosen field, not only in Minnesota and the Western Hemisphere, but in the World. He had the true Research Spirit and wrote considerably on the subjects of feeble-mindedness, e.g., a nine-page article on feeble-minded-

ness and criminality (1911). In his short work, entitled "Dwellers in the Vale of Siddim—A True Story of the Social Aspect of Feeble-mindedness,"‡ he follows one single feeble-minded family down through many generations; it reveals the many criminals and the huge sums in dollars, alone, this family cost the country (all the various States through which he traced them—a research that covered many years on his part). It is well-nigh unbelievable. If Dr. Arthur C. Rogers had not accomplished anything else worthwhile in his lifetime, this work, alone, would justify his having lived.

Rose, Francis M.—"A member of the firm of Rose & Wood, the leading physicians in this part of the state, was born in Columbus, Ohio, on March 25, 1841. Mr. Rose's grandfather visited the present site of that city in 1790, and located on a farm twelve miles south of there, where he died. Francis' father was a merchant in the same place, and remained there until his death. Dr. Rose attended the schools of his native place and the State University two years, commenced the study of medicine when sixteen years old at Starling Medical College and graduated three years later (1860). He practiced at West Rushville, Ohio, until 1861, when he was appointed Assistant Surgeon of the Forty-third Ohio Regiment; in April, 1862, was promoted to Regimental Surgeon, and in the fall to Division Surgeon, being then only twenty-one years old. In 1863, he was Medical Inspector of the Seventeenth Army Corps, on the staff of General Frank Blair; in 1864, was Inspector of General M. F. Forath's division, and remained until the close of the war. He then located in Ottawa, Illinois, and in 1868, removed to Faribault, forming the present partnership in 1874. On the 25th of November, 1876, Miss Cornelia W. Whipple became his wife. They have one child, Francis M." *(History of Rice County* by Rev. Edward D. Neill, 1882).

Dr. Rose was physician of St. Mary's Hall and the State School for the Blind.

Schmidt, Andrew J.—Practiced for a short time in Northfield for a few years. He is listed there from 1895 to 1898. Dr. Schmidt was a graduate of McGill University in 1886 and was licensed in Minnesota December 20, 1886. He died in November, 1902.

Scofield, John L.—The first physician to practice in Northfield, Dr. Scofield's biography appears out of alphabetic order, as the second in this series (Page 56, January, 1956 number).

Scott, M. W.—Practiced for a short time in Rice County. He had practiced in Morristown until 1870 and then located for one year in New Ulm. His fees were 25 cents in town, 50 cents in the country.§ He is probably identical with **Dr. Milo Wakely Scott** who graduated from Rush Medical College, Chicago, in 1877 and was licensed in Minnesota by exemption. He claimed that he had been in practice in Minnesota since 1868. During the 1880's he moved to Grand Forks where he is listed in the medical directories from 1886 through 1896. He was county physician and coroner and was a member of the first Capitol Commission which located the State Capitol at Bismarck. During the first years after the turn of the century he practiced in Minneapolis and later moved to California. He died at Los Angeles on September 4, 1910.

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‡Boston, 1919, 80 pages.

§Gorge B. Weiser, "History of Medicine in Brown County," MINNESOTA MEDICINE, p. 187, March, 1941.

Seeley, John Sanford.—He was born at Carlysle, N. Y., on June 12, 1852, to Isaac Seeley and Mary Fraser, both natives of the State of New York. He attended the public school at Saduo Bay, N. Y., and received his higher education at Carlysle Academy, Carlysle, N. Y. He started the study of medicine by reading in the office of Dr. Roscoe (or Rusco) at Carlysle, N. Y., and which he continued for four years. After that he entered the Medical School of the University of Michigan from which he graduated with the degree of Doctor of Medicine in the spring of 1876. At the time he started reading medicine in the office of Dr. Roscoe of Carlysle, he took to the lecture platform to become an advocate of local option. By the time he was eighteen years of age he was known throughout New York State as the "boy lecturer on the liquor evil." Illustrating his lectures with lantern slides, the young man traveled from town to town arousing sentiment against drunkenness and preaching sobriety throughout the length and breadth of his native state. The idealistic youth was an ardent and real reformer. Each summer during his several years of study at Ann Arbor, the young medical student resumed his anti-liquor lectures in the State of New York. By these net earnings he made enough money to pay for most of his medical education.

Following his graduation as an M.D., the young Dr. Seeley went to Farmington, Minnesota, where he hung out his shingle and lived with his paternal uncle, Nathan Seeley. While starting his practice, he worked as an ordinary farmhand in the harvest fields the summer and fall of his first year in Farmington. He went to Meeker County, but soon returned to Farmington to continue with his budding practice there. He married Miss Jennie Flood of Morristown, Minnesota, at Cordova, Minnesota, where he had removed to practice, in 1878, two years after he had first opened his office in Farmington. He practiced four years at Cordova. Again, he removed to Morristown, where he practiced for three years. In the year 1886, he made his final move to settle down to his permanent practice at Faribault, Minnesota.

Dr. John Sanford Seeley was licensed to practice medicine and surgery in the State of Minnesota, on April 15, 1884. He was also licensed in the State of Kansas and Washington. Including his medical apprenticeship under the tutelage of Dr. Roscoe, his association with the medical profession covered a period of well over fifty years. He died suddenly on Sunday morning, March 9, 1924, while in the act of dressing. The cause of his sudden death was either coronary or cerebral. He died within a few minutes following the onset, and before the arrival of the first M.D., Dr. Charles M. Robilliard of Faribault, to reach his deathbed. He was in his seventy-second year at the time of his death and had practiced in Faribault for thirty-eight years. He had suffered a mild paralytic stroke on February 22, 1924. From that time until his sudden death, he greeted those who visited him with a smile and chatted freely with them. (His speech was not affected by his stroke). He was also able to walk about the room all by himself. Dr. Seeley was active in Masonic circles, and was the most active of the original sponsors of the local De Molay. His daughter, Laura I. Seeley, M.D., resides at Long Beach, California, and his son, I. F. Seeley, M.D., at Tucson, Arizona.

The writer had the privilege of knowing Dr. Seeley during the last six years of his life. The picture of him the most clearly fixed in the writer's memory is that of the doctor in his Prince Albert coat, high-crowned silk hat, and swinging his cane (which he never carried except on Sundays, certain holidays, and on formal occasions), parading up and down Faribault's Main Street on nice Sunday mornings of late spring, summer, and early fall with his lady clinging to the doctor's left arm. Everybody in town and throughout all the surrounding countryside knew who Dr. Seeley was. He was an individualist with certain eccentricities of speech and manner that made him stand out amongst his fellows and which stamped the memory of his

distinctive personality, peculiarly all his own, on the minds of all who knew him. He was one in a million, who will not soon be forgotten.

Skeels, A. P.—(1829-1872) He graduated from the Homeopathic Medical College of Pennsylvania in 1867. He went west to St. Louis where he became professor of Materia Medica in the old Homeopathic Medical College of Missouri. While there he was editor of the *Western Homeopathic Observer*. He came to Northfield in January, 1870, suffering from pulmonary tuberculosis and he died there on May 16, 1872. He is said to have been the preceptor of Henry Hutchinson (1849-1910) of Northfield and St. Paul.*

Smith, Patrick Albert.—He was born July 27, 1866, at North Nation Mills, Province of Quebec, Canada, the son of Josiah and Mary (O'Brien) Smith. The family moved to Alfred, Ontario, and later to Papineauville, Province of Alberta, where he attended the English school. Following elementary work received in the Canadian public schools, he continued his education at higher institutions of learning. He graduated from the University of Ottawa in 1884, and was granted his M.D. degree by Jefferson Medical College, Philadelphia in 1893. He was married at Montreal, Canada, to Miss Lilias H. Brady of Montebello, P.Q., Canada, on April 20, 1892. She preceded him in death on March 4, 1916.

Dr. Smith began his career as a physician in Minnesota, at Dundas, soon after his graduation in Philadelphia, in 1893. He moved to Faribault in 1895. In 1910, he and Rev. Pere Monge, then pastor of the Sacred Heart church in Faribault, went to Europe together. Dr. Smith did some post-graduate work in London, Paris, and Vienna. While in Vienna he was elected a member of the Vienna branch of the American Medical Association. During his forty years' residence in Faribault, Dr. Smith played a prominent role in the community's political and professional life. In 1917 he was a candidate for mayor of Faribault. On February 22, 1922, he was married to Miss Loretta Lieb, her death occurring on October 7, of the same year. Dr. Smith served as Rice County coroner for several years. He resigned that position in 1928 to become a candidate for county commissioner. Following his election, he served continuously up to the time of his death on November 27, 1935.

For three years, from the destruction of the old courthouse by fire on February 25, 1931, to the dedication of the beautiful new building, about as fireproof as it could be built, on December 22, 1934, he devoted so much of his time, energetically and tirelessly, that he had to sacrifice much of his big practice to assure the erection of the new structure. He successfully won over all obstacles, including litigation, labor troubles, and factional disputes. Dr. Patrick Albert Smith is the one outstanding man responsible for Rice County's magnificient new courthouse, one of the finest in the State of Minnesota. It is a befitting monument to his fighting Irish heart. Old friends and patients, many of whom he had shown charity into the fourth generation, having brought several thousands of them into the world, the poor served as cheerfully and without remuneration in money as those better off, contributed to the severe coronary insult (a second or third) that resulted in his death. Professionally, the perfect gentleman was held in high esteem by his professional brothers, friends, and the public.

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*This information is taken from James Eckman, "Homeopathic and Eclectic Medicine in Minnesota," MINNESOTA MEDICINE, p. 868, October, 1941.

(To be concluded in October issue)

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul 2, Minnesota

F. H. Magney, M.D., Secretary

PHYSICIANS LICENSED FEBRUARY 4, 1955

January, 1955, Examination

Name	*School*			*Address*
BALZ, George Pickering	U. of Illinois	MD	1952	200 1st St., S.W., Rochester, Minn.
BASCOM, John Upton	Northwestern U.	MD	1953	Mpls. Gen. Hospital, Minneapolis, Minn.
BRENNAN, Joseph George	U. of Bologna Italy	MD	1947	407 Granite Exchange, St. Cloud, Minn.
CHANDLER, Howard Christy	Emory U.	MD	1951	U. of Minn. Hospitals, Minneapolis, Minn.
COTE, Jacques	Laval U.	MD	1951	200 1st St., S.W., Rochester, Minn.
DOBLER, Manfred	Eberhard-Karls U. Germany	MD	1951	829 Payne Ave., St. Paul, Minn.
ERICKSEN, Dean Marshall	U. of Illinois	MD	1954	Veterans' Adm. Hosp., Minneapolis, Minn.
HENRIE, John Nathanial	U. of Utah	MD	1952	200 1st St., S.W., Rochester, Minn.
HUGHES, Lewis Edwin	U. of California	MD	1953	314 E. Grand Ave., Eau Claire, Wis.
JANNACH, Joseph Robert	U. of Minnesota	MD	1954	3250 35th Ave. S., Minneapolis, Minn.
JAUNISKIS, Ruta Orlauskaite	U. of Kaunas, Lithuania	"Physician"	1942	Mounds Park Hospital, St. Paul, Minn.
LEGER, Francois	U. of Montreal	MD	1952	200 1st St., S.W., Rochester, Minn.
MAIER, Edwin Robert	U. of Kansas	MD	1943	200 1st St., S.W., Rochester, Minn.
MOFFITT, Emerson Amos	Dalhousie U.	MD	1951	200 1st St., S.W., Rochester, Minn.
NAKAS, Osvaldas	U. of Dusseldorf Germany	"Physician"	1948	State Hospital, Ionia, Michigan
NELSON, John Douglas	U. of Minnesota	MD	1954	401 N. 40th Ave. W., Duluth, Minn.
NESS, Richard Alton	U. of Minnesota	MD	1954	U. of Minn. Hospitals, Minneapolis, Minn.
PAULSEN, Elsa Caroline Proehl	U. of Minnesota	MD	1954	U. of Minn. Hospitals, Minneapolis, Minn.
PECK, Owen Calvin	Kansas U.	MD	1951	200 1st St., S.W., Rochester, Minn.
PETERSON, Lowell Frederick Albert	Northwestern U.	MD	1951	200 1st St., S.W., Rochester, Minn.
REDDING, Foster Kinyon	U. of Pennsylvania	MD	1954	4119 E. Lake St., Minneapolis, Minn.
RODIN, Ernst	U. of Vienna Austria	MD	1949	U. of Michigan Hospital, Ann Arbor, Mich.
RUTH, Bradley Robert	U. of Minnesota	MD	1952	818 19th Ave. E., Duluth, Minn.
SAGER, John Harmer	Geo. Washington U.	MD	1952	200 1st St., S.W., Rochester, Minn.
SELCK, Wolfgang Wilhelm Theodor Erich	U. of Hamburg Germany	MD	1952	2703 E. Lake St., Minneapolis, Minn.
SKWORCOW, George	U. of Heidelberg, Germany	MD	1952	545 Capitol Blvd., St. Paul, Minn.
VERNIER, Robert L.	U. of Cincinnati	MD	1952	U. of Minn. Hospitals, Minneapolis, Minn.
WEEKS, Byron Thomas	U. of Utah	MD	1951	Veterans' Adm. Hosp., Minneapolis, Minn.
WILLIAMS, Richard Eugene	St. Louis U.	MD	1954	Mpls. Gen. Hospital, Minneapolis, Minn.

Reciprocity Candidates

Name	*School*			*Address*
BURTON, Truman Young	U. of Utah	MD	1950	200 1st St., S.W., Rochester, Minn.
CHRISTIANSON, Mary Helen Faubion	U. of Texas	MD	1953	200 1st St., S.W., Rochester, Minn.
CASSEL, William Gilbert	Western Reserve	MD	1951	200 1st St., S.W., Rochester, Minn.
CLEMENTS, Betty Grace Elizabeth	U. of Nebraska	MD	1952	200 1st St., S.W., Rochester, Minn.
GRIFFITHS, Edward Krohn	U. of Pittsburgh	MD	1953	200 1st St., S.W., Rochester, Minn.
GRIZZLE, Claude Oliver	Stanford U.	MD	1954	200 1st St., S.W., Rochester, Minn.
HAMSA, William Rudolph	U. of Nebraska	MD	1929	527 Medical Arts Bldg., Omaha, Neb.
HICKS, JR., Wm. Wynne	Jefferson Med. Coll.	MD	1953	200 1st St., S.W., Rochester, Minn.
HIGGINS, Dennis Curran	Loyola U.	MD	1953	200 1st St., S.W., Rochester, Minn.
HILDYARD, Victor Herbert	U. of Kansas	MD	1942	Mpls. Gen. Hospital, Minneapolis, Minn.
HUNT, JR., Oliver Raymond	U. of Louisville	MD	1951	200 1st St., S.W., Rochester, Minn.
JARVIS, JR., Walter Hearn	U. of Texas	MD	1942	200 1st St., S.W., Rochester Minn.
KEOWN, JR., John Thomas	U. of Oklahoma	MD	1950	Pender, Nebr.
LANGRALL, JR., Harrison Morton	U. of Maryland	MD	1953	200 1st St., S.W., Rochester Minn.
LINDQUIST, Richard Harmon	U. of Louisville	MD	1947	200 1st St., S.W., Rochester Minn.
MARTIN, Jr., Peter Vincent	Hahnemann Med. Coll.	MD	1947	Prudential Ins. Co., Minneapolis, Minn.
MILES, JR., James Verner	U. of Pennsylvania	MD	1947	200 1st St., S.W., Rochester Minn.
PONTIUS, John Gilmore	U. of Pennsylvania	MD	1953	200 1st St., S.W., Rochester Minn.
RAYNAK, Frank Raymond	Temple U.	MD	1947	200 1st St., S.W., Rochester Minn.
RIBISI, Albert Prescott	Loyola U.	MD	1948	200 1st St., S.W., Rochester Minn.
ROBERTS, Frank Edwin	U. of So. California	MD	1954	17600 Chase St., Northridge, Calif.
SCALLIN, James Francis	U. of Michigan	MD	1953	U. of Minn. Hospitals, Minneapolis, Minn.
SHERANIAN, Lincoln Oscar	Stanford U.	MD	1954	200 1st St., S.W., Rochester Minn.
TOBIAN, JR., Louis	Harvard U.	MD	1943	U. of Minn. School of Med., Minneapolis, Minn.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

National Board Candidates

Name	*School*		*Address*
BATT, Bernard	N. Y. Med. Coll.	MD 1949	Presbyterian Hospital, N. Y., N. Y.
BURKE, Barbara Aune	Woman's Med. Coll., Pa.	MD. 1953	U. of Minn. Hospitals, Minneapolis, Minn.
CARNEVALI, John Francis	Georgetown U.	MD 1953	200 1st St., S.W., Rochester Minn.
CLEMETT, Arthur Reid	U. of Rochester	MD 1950	U. of Minn. Hospitals, Minneapolis, Minn.
COFFEY, Wm. Francis Xavier	Jefferson Med. Coll.	MD 1953	6606 Woodcrest Ave., Philadelphia, Pa.
DREWRY, Garth Richard	Harvard U.	MD 1952	U. of Minn. Hospitals, Minneapolis, Minn.
EICHENLAUB, John Ellis	Johns Hopkins	MD 1945	U. of Minn., Students Health Service, Minneapolis, Minn.
EVANS, Harold Warren	Cornell U.	MD 1949	200 1st St., S.W., Rochester Minn.
FAIRBAIRN, II, John Fitzgerald	U. of Buffalo	MD 1945	1132 Delaware Ave., Buffalo, N. Y.
GARDNER, Gawinn Bennion	U. of Utah	MD 1952	200 1st St., S.W., Rochester Minn.
GARDNER, John Underhill	N. Y. Med. Coll.	MD 1953	200 1st St., S.W., Rochester Minn.
GLUECK, Bernard Charles	Harvard U.	MD 1938	1441 E. River Rd., Minneapolis, Minn.
GOETZ, Frederick Charles	Harvard U.	MD 1946	U. of Minn. Hospitals, Minneapolis, Minn.
JOHNSON, Norman Paul	State U. of Iowa	MD 1948	329 W. 15th St., Minneapolis, Minn.
LEE, Robert Eugene	U. of Illinois	MD 1951	200 1st St., S.W., Rochester Minn.
LYONS, William Stewart	Geo. Washington U.	MD 1951	200 1st St., S.W., Rochester Minn.
MEIER, Donald Walter	U. of Rochester	MD 1950	200 1st St., S.W., Rochester Minn.
MESSER, James Walter	Johns Hopkins U.	MD 1953	200 1st St., S.W., Rochester Minn.
MOSKOWITZ, Roland Wallace	Temple U.	MD 1953	200 1st St., S.W., Rochester Minn.
MYERS, John William	U. of Iowa	MD 1953	103 W. 1st St., Canby, Minn.
OBERT, Mary Newton	Temple U.	MD 1950	915 3rd Ave., S.E., Rochester, Minn.
ROBINSON, Hugh Payson	U. of Rochester	MD 1953	200 1st St., S.W., Rochester Minn.
SHEA, Daniel Wright	St. Louis U.	MD 1951	200 1st St., S.W., Rochester Minn.
WATTENBERG, Lee Wolff	U. of Minnesota	MD 1950	U. of Minn. School of Med., Minneapolis, Minn.
WORRELL, Janet	Woman's Med. Coll.	MD 1953	200 1st St., S.W., Rochester Minn.
WORTHINGTON, JR., John Willian	Duke U.	MD 1951	200 1st St., S.W., Rochester Minn.

PHYSICIANS LICENSED MAY 6, 1955

April, 1955, Examination

Name	*School*		*Address*
ALTON, Donald Gilman	U. of Rochester	MD 1953	Box 425, Bird Island, Minn.
CAREY, Beverly Jane Baxter	Queen's U.	MD 1951	Rochester State Hosp., Rochester, Minn.
DEUTSCH, Robert John	Marquette U.	MD 1954	4831½ W. 5th St., Duluth, Minn.
EPP, Dennis Lee	Northwestern U.	MD 1954	517 Popular Ave., Philadelphia, Miss.
FEDDERS, Gerhard	U. of Youryeff, Russia	MD 1913	State Hospital, Anoka, Minn.
GOODLIN, Robert Clair	U. of Oregon	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
HALVERSON, Donald Eugene	U. of Minnesota	MD 1954	Winnebago, Minn.
HAMEL, Arnold Louis	Marquette U.	MD 1954	675 Water St., Excelsior, Minn.
HATFIELD, Clinton Bruce	U. of Alberta	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
HILL, Norman Charles	U. of Manitoba	MD 1952	200 1st St., S.W., Rochester Minn.
JENSEN, John Arnold	U. of Minnesota	MD 1954	119 N. Broadway, Crookston, Minn.
LARSON, Wyllis Gene	U. of Washington	MD 1954	111 E. 4th St., Northfield, Minn.
LOFSTROM, Dennis Earl	U. of Minnesota	MD 1954	Pine River, Minn.
MERIE, Josephine Nancy	McGill U.	MD 1953	200 1st St., S.W., Rochester Minn.
MULLEN, Sanford Allen	Columbia U.	MD 1949	U. of Minn. Hospitals, Minneapolis, Minn.
NELSON, Ralph Alfred	U. of Minnesota	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
RAAEN, Olaf Juel	U. of Oslo, Norway	MD 1939	214 Wesley Temple Bldg., Minneapolis, Minn.
SEBRECHTS, Paul	U. of Louvain, Belgium	MD 1946	515 18th Ave., S.W., Rochester, Minn.
SLETTEN, Ivan Wayne	U. of Wisconsin	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
SOWELL, Richard Chester	U. of Oregon	MD 1954	11207 S.E. Harold St., Portland, Ore.
TAYLOR, Keith Alan	U. of Oregon	MD 1954	500 Eastman Bldg., Boise, Idaho
THOMPSON, Russell Alden	U. of Minnesota	MD 1954	Professional Bldg., Cosmos, Minn.
WESTLEY, Kent Forbes	Temple U.	MD 1943	U. of Minn. Hospitals, Minneapolis, Minn.

Reciprocity Candidates

Name	*School*		*Address*
CHITTUM, John Raymond	Duke U.	MD 1953	East Range Clinic, Aurora, Minn.
DeBELLY, Carl George	U. of Nebraska	MD 1954	McGrew, Nebr.
EDWARDS, JR., Henry Grady	Med. Coll., Georgia	MD 1953	200 1st St., S.W., Rochester Minn.
GRAF, George Paul	U. of Wisconsin	MD 1950	200 1st St., S.W., Rochester Minn.
HANSEN, Robert Henry	Louisiana U.	MD 1942	200 1st St., S.W., Rochester, Minn.
KASPER, Robert Eugene	Marquette U.	MD 1949	U. of Minn. Hospitals, Minneapolis, Minn.
KRUEGER, Norman Leland	U. of Iowa	MD 1950	128 S. 76th St., Milwaukee, Wis.
KUYKENDALL, Samuel James	Vanderbilt U.	MD 1949	200 1st St., S.W., Rochester, Minn.
McCOWN, Louis Kenneth	Tulane U.	MD 1949	200 1st St., S.W., Rochester, Minn.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Name	*School*		*Address*
NELSON, Gunard Arvid	U. of So. California	MD 1954	200 1st St., S.W., Rochester, Minn.
NOLAN, Robert Kay	U. of Vermont	MD 1947	Western Montana Clinic, Missoula, Mont.
PETERMAN, Albert Frederick	Wash. U., St. Louis, Mo.	MD 1952	200 1st St., S.W., Rochester, Minn.
SAYCICH, John Michael	St. Louis U.	MD 1949	200 1st St., S.W., Rochester, Minn.
SWANSON, JR., Clarence Hilding	U. of Nebraska	MD 1954	Northwestern Hospital, Minneapolis, Minn.

National Board Candidates

Name	*School*		*Address*
DECKER, Barry	New York U.	MD 1952	200 1st St., S.W., Rochester, Minn.
LING, Victoria Kuo-fen	U. of Michigan	MD 1949	10410 E. Jefferson, Detroit, Mich.
REINER, Ernest August	Columbia U.	MD 1952	U. of Minn. Hospitals, Minneapolis, Minn.
ROTSTEIN, Jerome	State U. of N. Y.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
SCHAEFER, Joseph Charles	U. of Wisconsin	MD 1950	200 1st St., S.W., Rochester, Minn.
WILDER, Walter Llewellyn	Harvard U.	MD 1950	1801 Lyndale Ave. S., Minneapolis, Minn.

PHYSICIANS LICENSED JULY 8, 1955

June, 1955, Examination

Name	*School*		*Address*
ACKROYD, Albert Edwin	U. of Minnesota	MD 1955	San Diego Co. Gen. Hosp., San Diego, Calif.
ANDERSON, Oscar Donald	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
ANSELL, Julian Samuel	Tufts Med. Coll.	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.
AULICK, Ernest James	Coll. Med. Evang.	MD 1954	Belgrade, Minn.
BACKER, Gordon Lewis	U. of Minnesota	MD 1955	King Co. Hospital, Seattle, Wash.
BACKUS, Lloyd Benjamin	U. of Minnesota	MD 1955	Highland-Alameda Co. Hosp., Oakland, Calif.
BACKUS, Reno Eugene	U. of Minnesota	MD 1955	Highland-Alameda Co. Hosp., Oakland, Calif.
BECKETT, Peter Gordon Stewart	Trinity Col.., Ireland Bach. Med. & Surg. & Ob.	1945	951 E. LaFayette, Detroit, Mich.
BERG, Bruce Oscar	U. of Minnesota	MD 1955	Fitzsimons Army Hosp., Denver, Colo.
BERGERON, Dale A.	U. of Minnesota	MD 1955	Letterman Army Hosp., San Francisco, Calif.
BINGER, Charles Melvin	U. of Minnesota	MD 1955	Highland-Alameda Co. Hosp., Oakland, Calif.
BITTICK, Wilbur Howard	U. of Oregon	MD 1952	Mpls. Gen. Hospital, Minneapolis, Minn.
BLOCH, Henry Simon	U. of Minnesota	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
BLOOM, David	U. of Minnesota	MD 1955	King Co. Hospital, Brooklyn, N. Y.
BOEHLKE, Russell R.	U. of Minnesota	MD 1955	U.S.P.H.S. Hospital, Baltimore, M.D.
BOLANDER, John Ivar	U. of Minnesota	MD 1955	Vets' Adm. Hosp., Long Beach, Calif.
BOLT, Donald Arthur	U. of Minnesota	MD 1955	Walter Reed Army Hosp, Washington, D. C.
BRAINARD, John Brimhall	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
BRAZIE, Joseph V.	U. of Oregon	MD 1954	1/Lt. MC, USA, Armish/MAAG 8673 DU APO 205, N. Y., N. Y.
BUCKLEY, Robert Emmett	U. of Minnesota	MD 1955	So. Pacific RR Hosp., San Francisco, Calif.
BURNETT, George Hilgard	U. of Chicago	MD 1954	Service Mem. Inst., U. of Wis., Madison, Wis.
BUSHMAN, David E.	U. of Minnesota	MD 1955	St. Vincent's Hospital, Portland, Ore.
CALLEWART, Robert A.	U. of Minnesota	MD 1955	U. of Wisconsin Hosp., Madison, Wis.
CARLIN, Jean Effal	U. of Minnesota	MD 1954	U. of Minn. Hospitals, Minneapolis, Minn.
CARLSON, Homer John	U. of Minnesota	MD 1955	St. Luke's Hospital, Duluth, Minn.
CONNOLLY, Daniel Charles	University Coll., Ireland	MD 1948	200 1st St., S.W., Rochester, Minn.
CORNWELL, Samuel Owen	U. of Minnesota	MD 1955	Hillcrest Rd., Chapel Hill, N. C.
CORSER, David Hewson	Washington U (MO)	MD 1954	639 E. 18th St., Minneapolis, Minn.
COURTEAU, Robert David	U. of Minnesota	MD 1955	St. Mary's Hosp., Minneapolis, Minn.
COUTURE, Lorraine	U. of Manitoba	MD 1955	Baltimore City Hosp., Baltimore, Md.
DELANEY, John P.	U. of Minnesota	MD 1955	Santa Clara Co. Hosp., San Jose, Calif.
DION, Francis Richard	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
DUVALL, III, Arndt John	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
ELLINGSON, Richard Bennett	U. of Minnesota	MD 1955	4029 43rd Ave. S., Minneapolis, Minn.
ENGLUND, Garth Walfred	U. of Minnesota	MD 1955	Denver Gen. Hospital, Denver, Colo.
EPSTEIN, Wolfgang	U. of Minnesota	MD 1955	San Francisco Hosp., San Francisco, Calif.
FINK, Gerald Howard	U. of Minnesota	MD 1955	Mt. Sinai Hospital, Minneapolis, Minn.
FISH, George Herbert	U. of Minnesota	MD 1955	Virginia Mason Hosp., Seattle, Wash.
FRUCHTMAN, Stanley Arnold	U. of Minnesota	MD 1955	Mt. Sinai Hospital, Minneapolis, Minn.
GAMBLE, Charles Northam	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
GARETZ, Floyd Kenneth	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
GILLIS, Daniel Alexander	Dalhousie U.	MD 1953	200 1st St., S.W., Rochester, Minn.
GOEPFERT, Mary	U. of Minnesota	MD 1952	Veterans' Adm. Hosp., Minneapolis, Minn.
GORDON, Edwin Michael	U. of Minnesota	MD 1955	San Diego Co. Gen. Hosp., San Diego, Calif.
GORDON, John Ralph	Queen's U.	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.

Name	*School*		*Address*
GRIFFIN, Lee Martin	U. of Minnesota	MD 1955	U. S. Naval Hosp., Great Lakes, Ill.
GUSTAFSON, David Carl	Johns Hopkins	MD 1954	Robertsdale, Ala.
GUSTAFSON, Robert Warner	U. of Minnesota	MD 1955	St. Luke's Hospital, Duluth, Minn.
HALL, John Joseph	U. of Minnesota	MD 1955	St. Mary's Hospital, Minneapolis, Minn.
HALVERSON, Kermit John	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
HELWIG, JR., Karl Lyle	U. of Wisconsin	MD 1954	Kerkhoven, Minn.
HERRICK, Donald Willard	U. of Minnesota	MD 1955	St. Joseph's Hospital, St. Paul, Minn.
HILGEDICK, William Ralph	U. of Minnesota	MD 1955	St. Luke's Hospital, Duluth, Minn.
HOLM, Owen W.	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
HORNS, John Willard	U. of Minnesota	MD 1955	U. of California Hosp., Los Angeles, Calif.
HUSEBY, Thomas Lester	U. of Minnesota	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
HUSEBYE, Kjeld Oddvar	U. of Oslo, Norway	MD 1951	339 Lowry Medical Arts Bldg., St. Paul, Minn.
JACOBSON, Maynard Edward	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
JOHNSON, Lewis Allen	U. of Minnesota	MD 1955	So. Pacific Gen. Hosp., San Francisco, Calif.
JOHNSON, Ray Allen	U. of Minnesota	MD 1955	Highland-Alameda Co. Hosp., Oakland, Calif.
JORDHEIM, Robert Phillips	Northwestern U.	MD 1954	1507 A Sycamore, Big Spring, Texas
KANE, Dennis James	U. of Minnesota	MD 1955	U. of Minn. Hosp., Minneapolis, Minn.
KLEPP, Arne Leonard	U. of Minnesota	MD 1955	Vets' Adm. Hosp., Long Beach, Calif.
KNUDSON, Harris Willard	U. of Minnesota	MD 1955	Vets' Adm. Hosp., Long Beach, Calif.
KNUTSON, Kenneth Roger	U. of Minnesota	MD 1955	St. Luke's Hospital, Duluth, Minn.
KOOP, JR., Severin H.	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
KRUGER, JR., Elmer Lawrence	Med. Coll., Georgia	MD 1954	Mesaba Clinic, Hibbing, Minn.
KUNIN, Richard Allen	U. of Minnesota	MD 1955	New York Hospital, N. Y., N. Y.
LARSON, Theodore Gordon	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
LEAVITT, Clark Thomas	U. of Alberta	MD 1954	200 1st St., S.W., Rochester, Minn.
LEE, John Roland	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
LEONARD, Arnold S.	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
LEONARD, Stanley Alan	U. of Minnesota	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
LESSARD, Richard Joseph	U. of Minnesota	MD 1955	3303 N. Thomas Ave., Minneapolis, Minn.
LONEY, William Robert Ray	U. of Minnesota	MD 1955	Kansas City Gen. Hosp., Kansas City, Mo.
LOOFBOUROW, John Colm	U. of Minnesota	MD 1955	Gorgas Hospital, Ancon, Canal Zone
LYONS, Michael Murray	Loyola U.	MD 1948	U. of Minn. Hospitals, Minneapolis, Minn.
MARSCHKE, Gerald R.	U. of Minnesota	MD 1955	Vets' Adm. Hosp., Long Beach, Calif.
MARTIN, Donald Campbell	U. of Minnesota	MD 1955	Vets' Adm. Hosp., Long Beach, Calif.
MASSEE, Joseph Suter	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
MEYERS, Phillip Henry	U. of Minnesota	MD 1955	Kings Co. Hospital Center, Brooklyn, N. Y.
MORRIS, David Leslie	U. of Wisconsin	MD 1954	2302 Bruce Ave., Tucson, Ariz.
MOYER, Donald Glenn	U. of Minnesota	MD 1955	Letterman Army Gen. Hospital, San Francisco, Calif.
MURTAUGH, Robert Jean	U. of Minnesota	MD 1955	405 7th Ave. S.E., Minneapolis, Minn.
MUTCH, JR., Milton Gardner	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
NELDNER, Kenneth Herbert	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
O'KEEFE, Norbert John	U. of Minnesota	MD 1955	St. Joseph's Hospital, St. Paul, Minn.
OBERT, Gerard Joseph	U. of Pittsburgh	MD 1953	State Hospital, Rochester, Minn.
OLSEN, Jay Richard	Cornell U.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
OLSON, Lloyd Laures	U of Washington Seattle	MD 1954	Veterans' Adm. Hosp., Minneapolis, Minn.
OLSON, Robert William	U. of Minnesota	MD 1955	Orange Co. Gen. Hosp., Orange, Calif.
OSTERBERG, Kenneth Arvid	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
OWENS, Frank Joseph	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
PALMER, Karl Arthur	U. of Minnesota	MD 1955	City Hospital, Cleveland, Ohio
PETERSON, Raymond Dale August	U. of Minnesota	MD 1955	3633 45th Ave. S., Minneapolis, Minn.
PIERCE, Gordon Le Ray	U. of Minnesota	MD 1955	Kaiser Foundation Hosp., Oakland, Calif.
REMSBERG, John R. S.	U. of Minnesota	MD 1955	1846 Wellesley Ave., St. Paul, Minn.
REYNOLDS, Donald Parker	U. of Manitoba	MD 1948	Swedish Hospital, Minneapolis, Minn.
REYNOLDS, James Stanton	U. of Minnesota	MD 1955	Meadowbrook Hospital, Hempstead, N. Y.
RONDEAU, Eugene Thomas	U. of Minnesota	MD 1954	Hilltop House Apts., Wernersville, Pa.
ROSS, Howard David	U. of Minnesota	MD 1954	Vet. Adm. Center, Los Angeles, Calif.
ROTENBERG, Samuel	U. of Minnesota	MD 1955	Kings Co. Hospital Center, Brooklyn, N. Y.
RUNQUIST, Richard Keith	U. of Minnesota	MD 1954	Westbrook, Minn.
RYDELL, Robert Earl T.	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
SALOVICH, Edward Lamont	U. of Minnesota	MD 1955	Parkland Mem. Hospital, Dallas, Texas
SCHUFT, James Robert	U. of Minnesota	MD 1955	St. Mary's Hospital, Duluth, Minn.
SCHULZ, Robert Wilhelm	U. of Minnesota	MD 1955	St. Luke's Hospital, Duluth, Minn.
SEIFERT, Donald Ralph	Creighton U.	MD 1955	St. Mary's Hospital, Duluth, Minn.
SHANNON, Lloyd Wilburn	U. of Manitoba	MD 1946	Atlantic Coastline RR Hospital, Waycross, Ga.
SHELDON, Wilmer George	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
SHEPARD, Richard	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
SHORT, Marshall H.	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
SILVIS, Stephen Edward	U. of Minnesota	MD 1955	St. Joseph's Hospital, St. Paul, Minn.

Name	*School*		*Address*
SIMMONS, Richard Knute	U. of Minnesota	MD 1955	3939 Chowen Ave. S., Minneapolis, Minn.
SIPE, James Willard	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
SLUNGAARD, Rolv Kjelberg	U. of Oslo, Norway	MD 1950	3920 Sunnyside Ave., Minneapolis, Minn.
SMITH, Alan Garrigues	U. of Minnesota	MD 1955	D. C. Gen. Hospital, Washington, D. C.
SORKNESS, Paul O.	U. of Minnesota	MD 1955	Highland-Alameda Co. Hospital, Oakland, Calif.
STOLEN, Keith H.	U. of Minnesota	MD 1955	Tripler Army Hospital, Oahu, T. H.
STROBEL, Claire John Albert	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
SUBAK, Barbara Hanson	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
SWAIMAN, Kenneth Fred	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
SWAN, Harold James Charles	U. of London	1945	200 1st St., S.W., Rochester, Minn.
	Bach. Med. & Surg.		
SWANSON, Ralph Herbert	U. of Minnesota	MD 1955	Mercy Hospital, Toledo, Ohio
SWENSEN, Don Philip	U. of Minnesota	MD 1955	St. Luke's Hospital, Duluth, Minn.
SWENSON, JR., Richard William	U. of Minnesota	MD 1955	Hibbing, Minn.
SYVERSON, Leslie Arthur	U. of Minnesota	MD 1955	St. Mary's Hospital, Duluth, Minn.
THOMFORD, Neil Roger	U. of Minnesota	MD 1955	Highland-Alameda Co. Hospital, Oakland, Calif.
THOUIN, Laurence Gonzague	U. of Minnesota	MD 1943	2229 3rd Ave. E., Hibbing, Minn.
TRUPPMAN, Edward S.	U. of Minnesota	MD 1955	Jackson Mem. Hospital, Miami, Fla.
TVETEN, Omar A.	U. of Minnesota	MD 1955	Highland-Alameda Co. Hospital, Oakland, Calif.
VAALER, Robert T.	U. of Minnesota	MD 1955	Parkland Mem. Hospital, Dallas, Texas
VALASEK, Frances Elizabeth	U. of Minnesota	MD 1955	Sterling-Winthrop Res. Institute, Rensselaer, N. Y.
VAUBEL, Carol Lynn	U. of Minnesota	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
VANDERPOOL, Thomas Earl	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
WEATHERHEAD, Donald Stuart Philip	U. of Manitoba	MD 1948	U. of Minn. Hospitals, Minneapolis, Minn.
WHITE, James George	U. of Minnesota	MD 1955	Receiving Hospital, Detroit, Mich.
WILLIAMS, Francis Richard	U. of Minnesota	MD 1955	Presbyterian Hospital, Chicago, Ill.
WILSON, Robert K.	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.
WITTER, Robert LaMotte	U. of Minnesota	MD 1955	Mt. Sinai Hospital, Minneapolis, Minn.
WOOD, Lloyd Thomas	U. of Minnesota	MD 1954	Box 465, Forest Lake, Minn.
WOOLFREY, Bertram Ford	U. of Minnesota	MD 1955	Santa Clara Co. Hospital, San Jose, Calif.
YODER, Richard Dale	U. of Minnesota	MD 1955	Meadowbrook Hospital, Hempstead, N. Y.
ZELICKSON, Alvin S.	U. of Minnesota	MD 1955	Kings Co. Hospital, Brooklyn, N. Y.
ZINN, Charles W.	U. of Minnesota	MD 1955	Ancker Hospital, St. Paul, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BYRNE, William Joseph	St. Louis U.	MD 1954	Browerville, Minn.
CAUBLE, Charles Franklin	U. of Arkansas	MD 1949	214 Wesley Temple Bldg., Minneapolis, Minn.
ERICKSON, Raymond Leroy	U. of Oregon	MD 1944	852 Lowry Medical Arts Bldg., St. Paul, Minn.
FISHER, Albert Lee	U. of Wisconsin	MD 1950	Veterans' Adm. Hosp., Minneapolis, Minn.
HERRMAN, John Clinton	Jefferson Med. Coll.	MD 1953	200 1st St., S.W., Rochester, Minn.
INDECK, Walter	U. of Illinois	MD 1947	4959 Excelsior Blvd., Minneapolis, Minn.
KIOLBASA, Edward Benedict	U. of Nebraska	MD 1954	105 S. Main St., Stillwater, Minn.
PECK, Willard Raymond	U. of Nebraska	MD 1941	214 Wesley Temple Bldg., Minneapolis, Minn.
PETERSON, Alvin Charles	U. of Nebraska	MD 1953	125 W. Union St., Mora, Minn.
PHILLIPS, William McLeskey	U. of Tennessee	MD 1952	200 1st St., S.W., Rochester, Minn.
SULLIVAN, JR., William Albert	Tulane U.	MD 1947	U. of Minn. Hospitals, Minneapolis, Minn.
WATKINS, Charles William	St. Louis U., Mo.	MD 1951	200 1st St., S.W., Rochester, Minn.
WOOD, Matthew William	U. of Tennessee	MD 1950	200 1st St., S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
ALPERT, Abraham Edward	Creighton U.	MD 1949	Memorial Hospital, Virginia, Minn.
EFTELAND, Myles Elroy	Northwestern U.	MD 1954	Karlstad, Minn.
FIFER, Ellen Zinsser	Columbia U.	MD 1949	Student Health, U. of Minn, Minneapolis, Minn.
GLENCHUR, Harry	U. of Rochester	MD 1953	Veterans' Adm. Hosp., Minneapolis, Minn.
KRISTENSEN, Lowell Andrew	Yale U.	MD 1954	Chetek, Wis.
MOWLEM, Albert	Yale U.	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.
SASANO, JR., Joseph Richard	Georgetown U.	MD 1954	1843 Lurting Ave., N. Y., N. Y.
SIMENSTAD, John Otis	Johns Hopkins	MD 1954	Osceola, Wis.
TSUJI, Harold Kazuyoshi	Temple U.	MD 1952	517 Maple Lane, Edgsworth, Sewickly, Pa.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

PHYSICIANS LICENSED NOVEMBER 4, 1955

Name	*School*		*Address*
ANDERSON, Charles Clyde	U. of Illinois	MD 1954	968 40th St., Moline, Ill.
BOUCHER, Donald Lester	U. of Minnesota	MD 1953	2841 44th Ave. S., Minneapolis, Minn.
BUCK, Conrad Charles LeRoy	U. of West. Ontario	MD 1952	200 1st St., S.W., Rochester, Minn.
DEGALLIER, Daniel	U. of Lausanne, Switzerland	MD 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
DUNITZ, Norman	State U. of Iowa	MD 1953	200 1st St., S.W., Rochester, Minn.
EITZMAN, Donald Vern	U. of Iowa	MD 1954	U. of Minn. Hospitals, Minneapolis, Minn.
FAILOR, Harlan John	U. of Wisconsin	MD 1954	200 1st St., S.W., Rochester, Minn.
FEDDERS, Skaidrite	U. of Latvia	1940 "Physician"	U. of Minn. Hospitals, Minneapolis, Minn.
FOERSTER, Darryl Kemp	U. of Toronto	MD 1951	200 1st St., S.W., Rochester, Minn.
FROST, Donald Morgan	Northwestern U.	MD 1955	Miller Hospital, St. Paul, Minn.
GREEN, Leland James	U. of Minnesota	MD 1955	Mt. Sinai Hospital, Minneapolis, Minn.
HASSEL, JR., Carl Walter	Jefferson Med. Coll.	MD 1954	200 1st St., S.W., Rochester, Minn.
JOHNSON, Paul Einar	U. of Minnesota	MD 1955	St. Mary's Hospital, Duluth, Minn.
LEHOUX, Louis Marie	Laval U.	MD 1953	200 1st St., S.W., Rochester, Minn.
MACH, John Richard	Marquette U.	MD 1955	St. Mary's Hospital, Minneapolis, Minn.
McCLARY, JR., Geo. Brewer	Northwestern U.	MD 1953	200 1st St., S.W., Rochester, Minn.
McRAE, Laird Donald	Northwestern U.	MD 1955	St. Mary's Hospital, Duluth, Minn.
O'ROURKE, William James	Creighton U.	MD 1955	St. Mary's Hospital, Duluth, Minn.
PETERS, Edward Hilliard	Northwestern U.	MD 1955	St. Mary's Hospital, Duluth, Minn.
STERN, Marvin Lee	U. of Manitoba	MD 1955	Mt. Sinai Hospital, Minneapolis, Minn.
TRAPNELL, Richard Henry	U. of Pennsylvania	MD 1952	200 1st St., S.W., Rochester, Minn.
VINES, Benjamin Glenn	Howard U.	MD 1954	Veterans' Adm. Hosp., Minneapolis, Minn.
WESTMAN, Lincoln Herbert	U. of Wisconsin	MD 1953	200 1st St., S.W., Rochester, Minn.
WHITE, Robert Joseph	Harvard U.	MD 1953	200 1st St., S.W., Rochester, Minn.

Reciprocity Candidates

Name	*School*		*Address*
ANDERSON, Arden Oris	U. of Washington	MD 1954	Iron Exchange Bldg., Brainerd, Minn.
BORMAN, John Otto	Jefferson Med. Coll.	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.
COURTRIGHT, Claiborne Lee	U. of Oklahoma	MD 1951	200 1st St., S.W., Rochester, Minn.
ENGEL, Stephen	U. of Texas	MD 1954	200 1st St., S.W., Rochester, Minn.
FLATT, John R.	Northwestern U.	MD 1953	Community Clinic, Wabasha, Minn.
HARP, Robert Allen	U. of Texas	MD 1954	200 1st St., S.W., Rochester, Minn.
HATLEBERG, James Larrabee	Marquette U.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
HOLLAND, Robert Hughes	Jefferson Med. Coll.	MD 1944	Veterans' Adm. Hospital, Dallas, Texas
HAZLEDINE, Herbert John	U. of Michigan	MD 1943	200 1st St., S.W., Rochester, Minn.
HOWLAND, JR., Willard J.	U. of Kansas	MD 1950	200 1st St., S.W., Rochester, Minn.
McDONNELL, Thomas Richard	Creighton U.	MD 1944	200 1st St., S.W., Rochester, Minn.
MOLLISON, Malcolm	U. of Oklahoma	MD 1946	1101 E. Pecan, Altus, Okla.
NEWNUM, Raymond Lavern	Indiana U.	MD 1951	200 1st St., S.W., Rochester, Minn.
PETSCH, Kenneth Ray	Wash. U. (Mo.)	MD 1945	45 N. Snelling Ave., St. Paul, Minn.
PLOTT, Carol LeVan	U. of Virginia	MD 1943	Heron Lake, Minn.
POLLOCK, JR., Anthony J.	Marquette U.	MD 1948	Vet. Adm. Regional Office, Minneapolis, Minn.
PROUD, Harry Stewart	U. of Oregon	MD 1950	No. Pacific Beneficial Hospital, St. Paul, Minn.
ROSS, Griff Terry	U. of Texas	MD 1945	200 1st St., S.W., Rochester, Minn.
SCOTT-MILLER, James Robert	U. of Nebraska	MD 1954	200 1st St., S.W., Rochester, Minn.
SMITH, William Randolph	U. of Tennessee	MD 1950	200 1st St., S.W., Rochester, Minn.
STEEL, Helen Jean	U. of Texas	MD 1954	200 1st St., S.W., Rochester, Minn.
THEYE, Richard Albert	Indiana U.	MD 1948	200 1st St., S.W., Rochester, Minn.
VIDAL-GILET, Jose H.	U. of Arkansas	MD 1949	U. of Minn. Hospitals, Minneapolis, Minn.
WILBUR, Ronald Eugene	U. of Kansas	MD 1946	200 1st St., S.W., Rochester, Minn.
YOSS, Robert Eugene	U. of Tennessee	MD 1948	200 1st St., S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
COTTONE, Francis John	Georgetown U.	MD 1954	200 1st St., S.W., Rochester, Minn.
FRANTZ, JR., Ivan De Ray	Harvard U.	MD 1941	U. of Minn. Hospitals, Minneapolis, Minn.
GLICK, Dallas Donald	U. of Chicago	MD 1954	200 1st St., S.W., Rochester, Minn.
GREENSPAN, Richard Henry	Syracuse U.	MD 1948	U. of Minn. Hospitals, Minneapolis, Minn.
HAYNES, JR., Vital E.	Creighton U.	MD 1952	200 1st St., S.W., Rochester, Minn.
HOFFMAN, David Leland	Cornell U.	MD 1945	200 1st St., S.W., Rochester, Minn.
HUNTER, Samuel Joseph	Yale U.	MD 1954	200 1st St., S.W., Rochester, Minn.
LEONARD, John Paul	Albany Med. Coll.	MD 1954	Veterans Adm. Hospital, Minneapolis, Minn.
MARQUIS, John William	Geo. Wash. U.	MD 1954	200 1st St., S.W., Rochester, Minn.
MARTIN, Maurice John	U. of Illinois	MD 1954	200 1st St., S.W., Rochester, Minn.
MAYER, Richard Frederick	U. of Buffalo	MD 1954	200 1st St., S.W., Rochester, Minn.
MINOR, Ralph Hugh	U. of Washington	MD 1954	200 1st St., S.W., Rochester, Minn.

Name	*School*		*Address*
MOORMAN, JR., Warren L.	Med. Coll. Va.	MD 1943	200 1st St., S.W., Rochester, Minn.
MORRELL, Frank	Columbia U.	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.
RAMSDELL, John Alan	Columbia U.	MD 1954	200 1st St., S.W., Rochester, Minn.
ROSENDORF, Stanley Bernard	Geo. Wash. U.	MD 1953	416 Florida Ave., NE., Wash., D. C.
STORMONT, James Russell	Geo. Wash. U.	MD 1954	200 1st St., S.W., Rochester, Minn.
VANCE, John William	U. of West Ontario	MD 1951	200 1st St., S.W., Rochester, Minn.
WALSH, John Joseph	N. Y. Med. Col.	MD 1953	200 1st St., S.W., Rochester, Minn.
WOODWARD, JR., Edward	Med. Col. Va.	MD 1951	200 1st St., S.W., Rochester, Minn.
YONEHIRO, Earl Grant	U. of Rochester	MD 1952	U. of Minn. Hospitals, Minneapolis, Minn.
YOUKER, James Edward	U. of Buffalo	MD 1954	U. of Minn. Hospitals, Minneapolis, Minn.
ZIEGLER, Dewey Kiper	Harvard U.	MD 1945	U. of Minn. Hospitals, Minneapolis, Minn.

PHYSICIAN'S LICENSE REVOKED FOLLOWING ABORTION CONVICTION

Re: Revocation of the License of William L. Minkowski, M.D.

On February 10, 1956, the Minnesota State Board of Medical Examiners revoked the medical license held by Dr. William L. Minkowski of Redwood City, California. Dr. Minkowski, who did not appear at the hearing on that date, had been charged in a citation issued by the Board with having been convicted of two felonies in the Superior Court of San Mateo County, California. The citation charged Dr. Minkowski with having been convicted on or about April 26, 1954, of the crimes of abortion and conspiracy to commit abortion. A certified copy of the proceedings of the Superior Court discloses that Dr. Minkowski was sentenced to a term of one year in the county jail of San Mateo County on each of the two charges, the sentences to run concurrently. The sentence also provided that in addition to serving the one-year term, Dr. Minkowski was to be on probation for two years.

Dr. Minkowski, who was born in Mt. Vernon, New York, on February 20, 1917, graduated from the University of Minnesota with an M.D. degree on December 16, 1943. He served an internship at Philadelphia General Hospital and then took postgraduate work at Beth Israel Hospital, New York City, from July, 1946, to June, 1947. Dr. Minkowski was licensed in Minnesota by examination on February 7, 1947.

MINNEAPOLIS QUACK SENTENCED FOR ILLEGAL HEALING

Re: State of Minnesota vs. Davis Stern, also known as David Stern and "Doc" Stern.

On May 22, 1956, Davis Stern, sixty-eight, a resident of the Sheridan Hotel, Minneapolis, entered a plea of guilty before the Hon. Harold N. Rogers, Judge of the District Court of Hennepin County, to an information charging him with practicing healing without having a certificate of registration in the basic sciences, whereupon Judge Rogers sentenced the defendant to a term of one year in the Hennepin County Jail. Judge Rogers stayed the execution of the sentence for a period of one year to allow Stern to return to the State of New York and he was ordered to turn over his medical books and instruments to Minneapolis General Hospital. The sentence was suspended on the further condition that Stern is to refrain from practicing healing in any form in any state and also from giving medical advice. Stern was charged with illegally practicing healing after it was learned that he had examined a Minneapolis man and diagnosed his case as high blood pressure, making a total charge of $12.95 for two types of medicine which he furnished to the patient.

When questioned by the Court, the defendant stated that he was born on September 17, 1887, in New York City, and although he admitted that he had never graduated from a medical school, Stern claimed that he had taken courses at a medical school in New York City in 1907. Stern admitted that he had various medical instruments in his room at his hotel and that he had seen patients there, but he denied having charged patients for diagnosis or treatment. When he was asked how he was able to live without making charges, the defendant replied that he received $98.50 per month in the form of a pension and also two $25.00 checks each month "from friends." Stern holds no license to practice any form of healing in Minnesota.

GRANITE FALLS "REFLEXOLOGIST" SENTENCED FOR ILLEGAL HEALING

Re: State of Minnesota vs. Percy Graham

On May 28, 1956, Percy Graham, sixty-six, Granite Falls, Minnesota, entered a plea of guilty before the Hon. C. A. Rolloff, Judge of the District Court at Olivia, Minnesota, to an information charging him with practicing healing without having a certificate of registration in the basic sciences and Judge Rolloff then sentenced Graham to serve one year in the County Jail of Chippewa County, which was the county in which the offense occurred. The execution of the sentence was stayed by the Court, however, and the defendant was placed on probation for one year on condition that he obeys all laws and the rules and regulations prescribed in connection with his probation.

After it was reported to the Minnesota State Board of Medical Examiners that Graham was engaged in healing activities, a representative of the Board investigated the matter and learned that a forty-two-year-old woman patient, who was hospitalized at the Granite Falls Municipal Hospital under the care of a doctor of medicine, had previously been taking treatments from the defendant for her condition. Graham made a charge of $2.50 for each visit to the patient and in addition was paid the sum of $60.00 for a "foot bath." According to the patient, Graham twisted her toes, rubbed her hands, legs, back, spine and head with his hands and at other times he rubbed her with one hand while he held an electric vibrator in the other hand. She also said that Graham advised her not to eat toast or oatmeal because it had charcoal in it.

While being questioned prior to being sentenced in court, Graham stated that he was born at Bird Island, Minnesota, on May 14, 1890, and that his education consisted of grade school and three years of high school. The defendant, who admitted that he was not licensed to practice any form of healing in any state, referred to his work as "reflexology." He claimed that he had taken one four-day course in this subject in Sioux Falls, South Dakota, in 1954 and another similar course in 1955. In describing "reflexology" Graham said that the nerve endings in the toes of the feet connect with the various organs of the body and therefore these organs are stimulated when those nerve endings are massaged. Graham admitted that he usually charged the patient from $2.50 to $3.00 per treatment.

Meetings and Announcements

MEDICAL MEETINGS

State

Minnesota Society of Obstetrics and Gynecology, fall meeting, Rochester, Minnesota, September 22. Dr. Edward A. Banner, secretary, Rochester, Minnesota.

National

American Association of Blood Banks, ninth annual meeting, Somerset Hotel, Boston, Massachusetts, September 3-5. Secretary Marjorie Saunders, 725 Doctors Building, 3707 Gaston Ave., Dallas, Texas.

American Heart Association, 29th annual scientific session, Cincinnati, Ohio, October 26-29. Write American Heart Association, 44 East 23rd Street, New York 10, New York.

Minnesota-Dakota-Manitoba Orthopedic Society, annual meeting, Red Wing, Minnesota, September 7 and 8. Dr. Hollis Ahlrin, secretary, Rapid City, South Dakota.

National Society for Crippled Children and Adults, annual convention, Hotel Statler, Washington, D. C., October 28-31.

Postgraduate Medical School of New York University—Bellevue Medical Center, eight-week course in occupational medicine, September 10-November 2. Write Dean, N.Y.U. Postgraduate Medical School, New York 16, New York.

University of Illinois Department of Otolaryngology, Annual Assembly in Otolaryngology, October 1-7. Write Department of Otolaryngology, 1853 West Polk Street, Chicago 12, Illinois.

West Virginia State Medical Association, 89th annual meeting, The Greenbrier, White Sulphur Springs, West Virginia, August 23-25.

International

Congress of International Society of Hematology, Hotel Somerset, Boston, Massachusetts, August 27-September 1. Dr. W. C. Moloney, secretary, 39 Bay State Road, Boston, Massachusetts.

European Congress of Cardiology, Stockholm, Sweden, September 10-14. Dr. Karl E. Grewin, secretary, Sodersjukhuset, Stockholm, Sweden.

Inter-American Congress of Cardiology, Havana, Cuba, November 4-10. Dr. Ramon Aixala, Apartado 2108, Havana, Cuba.

Inter-American Conference on Occupational Medicine and Toxicology, Miami, Florida, September 3-7. Sponsored by the schools of medicine of University of Miami and University of Havana, Cuba. General chairman, Dr. Homer F. Marsh, dean, School of Medicine, University of Miami.

International Congress of Clinical Chemistry, Hotel New Yorker, New York, September 9-14. Secretary, John G. Reinhold, 711 Maloney Building, University of Pennsylvania, Philadelphia 4, Pennsylvania.

International Congress of Internal Medicine, Madrid, Spain, September 19-23. Dr. J. C. DeOya, secretary, No. 90, Madrid, Spain.

International Congress of International College of Surgeons, Palmer House, Chicago, Illinois, September 9-13. Dr. Max Thorek, secretary general, 1516 Lake Shore Drive, Chicago, Illinois.

International Congress of Radiology, Mexico, D. F., Mexico, July 22-28. Dr. Jose Noriega, secretary, Tepic 126 (2 e piso), Mexico, D. F. 7, Mexico.

International Congress on Diseases of the Chest, Cologne, Germany, August 19-23. Murray Kornfeld, executive director, 112 East Chestnut St., Chicago 11, Illinois.

Pan American Congress on Cancer Cytology, Miami, Florida, January 8-12, 1957. Dr. J. Ernest Ayre, chairman, 1155 N. W. 14th St., Miami, Florida.

Pan-Pacific Surgical Association, seventh congress, Honolulu, Hawaii, November 14-22, 1957. Write Dr. F. J. Pinkerton, director-general of the Pan-Pacific Surgical Association, Room 230, Young Building, Honolulu, Hawaii.

Second World Conference on Medical Education, World Medical Association, Chicago, Illinois, August 30 to September 4, 1959.

World Medical Association, Havana, Cuba, October 9-15. Dr. Louis H. Bauer, secretary general, 345 East 46th St., New York 17, New York.

SPECIAL NOTICE

The dates for the 104th annual meeting of the Minnesota State Medical Association have been changed. The meeting, scheduled for St. Paul, will be held *May 13, 14 and 15, 1957,* instead of May 20, 21, 22 as previously announced.

INTERNATIONAL COLLEGE OF SURGEONS EXAMINATIONS

Examinations for qualified fellows of the International College of Surgeons will be held in Chicago, Illinois, July 23 and 24 and October 29 and 30. Oral conferences will be held on August 6 and October 22. For details, write Secretary of the Qualifications Council, International College of Surgeons, 1516 Lake Shore Drive, Chicago 10, Illinois.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY CERTIFICATION

Of a total of 415 candidates who participated in the Part II examinations of the American Board of Obstetrics and Gynecology last year, 317 were certified. Among them were the following Minnesota physicians: Dr. Arthur M. Antonow, Virginia; Dr. Erick Y. Hakanson, St. Paul; Dr. Morris S. Rothnem, Minneapolis; Dr. Francis M. Swain, Minneapolis, and Dr. Kenneth W. Teich, Duluth.

AMERICAN HEART ASSOCIATION ACCEPTING APPLICATIONS

Applications by research investigators for support of studies in or related to the cardiovascular field are now being accepted by the American Heart Association. The awards would be for the fiscal year beginning July 1, 1957. Application deadline for research fellowships and established investigatorships is September 15, 1956. For grants-in-aid the application deadline is November 1. Further information and application forms are available from the Medical Director, American Heart Association, 44 East 23rd Street, New York 10, New York.

MINNESOTA SURGICAL SOCIETY

At a meeting of the Minnesota Surgical Society in Rochester May 11, 1956, the following officers were elected for the coming year:

D. O. Ferris, M.D., Rochester, president; Logan Levin, M.D., St. Paul, vice-president; Owen G. McDonald, M.D., Duluth, secretary-treasurer.

AMERICAN UROLOGICAL ASSOCIATION AWARD

The American Urological Association offers an annual award of $1,000 (first prize of $500, second prize $300 and third prize $200) for essays on the result of some clinical or laboratory research in Urology. Competition shall be limited to urologists who have been graduated not more than ten years, and to hospital internes and residents doing research work in Urology.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Hotel William Penn, Pittsburgh, Pennsylvania, May 6-9, 1957.

For full particulars, write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. Essays must be in his hands before December 1, 1956.

In Memoriam

EDGAR A. KING

Dr. Edgar A. King, Minneapolis, died June 1, 1956. He was ninety-one years old.

A graduate of Chicago Homeopathic Medical College and of Rush Medical College, he began to practice medicine in 1909. He served as a Captain in the medical corps in World War I and was affiliated with the Health Department of the City of Minneapolis and was on the staffs of both St. Mary's and Northwestern Hospitals in Minneapolis.

Dr. King was a member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors include a daughter, Bessie, Minneapolis, and a son, Dr. Zerah P. King, Los Angeles, California.

AUGUST C. TINGDALE

Dr. August C. Tingdale, former superintendent of the Minneapolis workhouse, died June 5, 1956. He was eighty-three years old.

A Minneapolis physician and surgeon for more than fifty years, Dr. Tingdale was born in Norway, graduated from Hamline University Medical School, St. Paul, and studied surgery in Vienna, Austria.

He was on the staff of Deaconess Hospital, was physician for the Ebenezer Home and a member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He also belonged to the Scottish Rite, Masonic Lodge 224, Zuhrah Temple, Phi Rho Sigma medical fraternity and the Norwegian Lunch Club.

Survivors include a son Randolph, New York; a daughter, Mrs. Owen Heath, St. Louis Park, and four sister, Mrs. Olive Jenson and Mrs. Mary Hanson, St. Louis Park; Mrs. Katherine Marsh, New York, and Mrs. Sigrid Leland, Seattle, Washington.

ROBERT R. TRACHT

Dr. Robert R. Tracht, a member of the University of Minnesota Medical School faculty, died May 25, 1956. He was fifty-six years old.

Dr. Tracht was a clinical assistant professor and refractionist in the department of ophthalmology at the University. He was also a practicing ophthalmologist in St. Paul with offices in the Lowry Medical Arts Building.

A graduate of Whittenberg College, Springfield, Ohio, and of Loyola University Medical School, Chicago, he was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a fellow of the National Academy of Ophthalmology and Otolaryngology.

Survivors include his wife, Ann; two daughters, Sigrid and Catherine, and a son, Robert, all of St. Paul, and a sister, Mrs. Harold Sayles, Cleveland, Ohio.

Woman's Auxiliary

PRESIDENT'S MESSAGE

All of us who are concerned about auxiliary work are happy that every county in the state is now organized. We are freed of concern about forming new auxiliaries and securing members-at-large; we can concentrate on increasing our membership and making each of our thirty-four county units a live and vital organization.

Each auxiliary has specific needs. Small county groups wish to learn more about setting up committees to deal with material sent from state and national offices. Hennepin and Ramsey counties, realizing the need for closer personal relationship among doctors' wives in cities, have initiated neighborhood meetings: a move toward decentralization and informality. The problems are many and varied.

This year we had a membership increase of 221, about 11 per cent. There are 3,406 physicians in the state medical association and 2,182 auxiliary members. That looks as if about 1200 eligible doctors' wives see no merit in joining the auxiliary, but actually the number is closer to 700. There are groups of doctors whose wives would not likely be members: men in the armed services, men in residencies or fellowships, and the associate, affiliate, and life members. Several counties have 100 per cent membership and we congratulate them. We will continue to try for a 10 per cent increase in membership next year.

Regional meetings were held at Hibbing, Jackson, and Rochester. These meetings serve an excellent purpose in allowing neighboring auxiliaries to get better acquainted and discuss auxiliary affairs informally.

Good auxiliary work depends on close relations between the state board and county units. The county president is a member of the state board and is directly responsible for bringing to her auxiliary the directives of the state board. It is imperative that she or her delegate be present at board meetings. This relationship is the basis of the entire auxiliary organization. Board meetings and regional meetings are our channels for relaying information to the county and the individual.

Mrs. C. L. Oppegaard and I shall attend the conference for presidents and presidents-elect in Chicago, October 1-3. Our own Minnesota school of instruction will be held at the Curtis Hotel, Minneapolis, on October 9. All auxiliary members are welcome and all committee chairmen should be present for instruction and reports from the Chicago meeting. Attendance at this fall meeting is a MUST for committee chairmen and county presidents.

MRS. L. P. HOWELL
President

OFFICERS AND COMMITTEE CHAIRMEN 1956-1957

Officers

President—Mrs. L. P. Howell, Rochester
President-Elect—Mrs. C. L. Oppegaard, Crookston
First Vice President—Mrs. Reuben Erickson, Minneapolis
Second Vice President—Mrs. M. F. Fellows, Duluth
Third Vice President—Mrs. Wallace Ritchie, St. Paul
Fourth Vice President—Mrs. R. H. Wilson, Winona
Recording Secretary—Mrs. E. R. Hudec, Echo
Corresponding Secretary—Mrs. John M. Waugh, Rochester
Treasurer—Mrs. W. B. Stromme, Minneapolis
Auditor—Mrs. George Penn, Mankato
Historian—Mrs. H. E. Bakkila, Duluth
Parliamentarian—Mrs. Leo W. Fink, Minneapolis

Regional Advisors

First District—Mrs. Howard F. Polley, Rochester
Second District—Mrs. David Halpern, Brewster
Third District—Mrs. M. I. Hauge, Clarkfield
Fourth District—Mrs. Alton E. Lindblom, Mankato
Fifth District—Mrs. Harold Peterson, St. Paul
Sixth District—Mrs. L. Raymond Scherer, Minneapolis
Seventh District—Mrs. Leslie Evans, Sauk Rapids
Eighth District—Mrs. Robert Estrem, Fergus Falls
Ninth District—Mrs. Andrew Sinamark, Hibbing

Committee Chairmen

Standing Committees

Allied Medical Careers—Mrs. E. H. Soule, Rochester
A.M.E.F.—Mrs. W. E. Wellman, Rochester
Archives—Mrs. James L. Benepe, St. Paul
Bulletin—Mrs. Ralph Eckman, Duluth
Cancer—Mrs. John Cameron, Crookston
Editor (MINNESOTA MEDICINE)—Mrs. A. B. Rosenfield, Minneapolis
Finance—Mrs. Mark Ryan, St. Paul
Health Days—Mrs. Conrad Karleen, Minneapolis
Legislation—Mrs. W. P. Gardner, St. Paul
Medical and Surgical Relief—Mrs. W. T. Greenfield, Cokato
Newsletter: The Gopher Doctor's Wife—Mrs. C. E. Carlson, Alexandria
Organization—Mrs. C. L. Oppegaard, Crookston
Press and Publicity—Mrs. Leonard Arling, Minneapolis
Printing and Roster—Mrs. L. R. Boies, Hopkins
Program and Health Education—Mrs. Philip Arzt, St. Paul
Public Relations—Mrs. M. O. Wallace, Duluth
Resolutions—Mrs. W. A. Merritt, Rochester
Revisions—Mrs. Harold Wahlquist, Minneapolis
School of Instruction—Mrs. Karl Anderson, Excelsior
Today's Health—Mrs. H. P. Van Cleve, Austin

Special Committees

Civil Defense—Mrs. J. A. Cosgriff, Olivia
In Memoriam—Mrs. Charles Waas, St. Paul
Mental Health—Mrs. William Gjerde, Lake City